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DUODENAL ULCER COMPLICATIONS; CONTROVERSIAL PROBLEMS IN SURGICAL MANAGEMENT *

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Many controversial problems exist in surgical management of complicated duodenal ulcer. The ulcer material in Charity Hospital of Louisiana at New Orleans, from 1951 until the present, has been used as reference and results of that study will be presented as various surgical aspects of duodenal ulcer are discussed.

Indications for surgery are hemorrhage, perforation, and obstruction. One form of perforation is posterior penetration onto the pancreas, manifested by continuous pain which radiates through to the back and is not relieved by food and antacids. Penetration should be treated definitively exactly as any other perforation, and yet, it is likely to be handled conservatively (See Figure 1).

Not generally listed as an indication in duodenal ulcer is occasional confusion with malignancy which demands exploration. A large penetrating ulcer near the ampulla of Vater may simulate duodenal or pancreatic malignancy and a fixed posterior ulcer near a mobile pylorus cannot be distinguished at times from a pyloric channel ulcer having cancer possibilities. Seldom should refractoriness to routine therapy sway the surgeon to action in a patient whose chief dissatisfaction is with minor alterations in day to day existence imposed on him by restrictions of conventional ulcer treatment.

DUODENAL ULCER
1951-'53 CHARITY HOSPITAL

PATIENTS 623	DEATHS 40	MORTALITY 6.4% SURGERY
Uncomplicated Ulcer	170	35
Penetrated	33	13
Obstruction	93	65
Hemorrhage	229	93
Perforation	98 (7)	90

Figure 1. Column 1 denotes the number of patients. Column 2, the number having some type of gastric surgery.

These indications are also influenced by age. A more aggressive attitude has been adopted in patients under 20 and over 60 having complicated duodenal ulcer. Complications in the young portend many future difficulties, so much so that surgery has to be considered early in the course of the disease. No surgeon looks with favor on removing the stomach of a person at this time in life. However, to avoid enthreatening and invaliding developments of ulcer in the young and to restore the person to a state of health which allows full range of activity oftentimes calls for operative intervention. A person over 60 runs less risk from surgery than he does from an episode of severe hemorrhage, obstruction, or perforation

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and if he gets a complication, he is a surgical candidate.

Apparent to all of us is the gradual reduction of mortality from massive gastroduodenal hemorrhage as a result of improved medical management, largely due to giving blood to replace that which is lost, and sparing use of transfusion still advocated occasionally is to be decried. Even with an aggressive medical program (See Figure 2) deaths occur from ulcer

HEMORRHAGE FROM DUODENAL ULCER

	Number of Patients	DEATHS	MORTALITY RATE
TOTAL.....	229	21	9.2%
CONSERVATIVE..	136	12	8.8%
SURGICAL.....	93	9	9.7%
Emergency Gastrectomy	20	4	20.0%
Elective Gastrectomy	73	5	6.85%

Figure 2

hemorrhage. The knotty problem is to pick out a small group in which the risk of conservative treatment is exorbitantly high. Certain factors may be guides, the patient over 45, in shock, losing bright red blood, bleeding persistently or repeatedly or having severe pain.

Ulcer hemorrhage remains without a clear cut indication for decisive surgical action. In massive hemorrhage the decision must rest on the evaluation of response to therapy, an index to continued hemorrhage. In the older age group, six to twenty-four hours may be sufficiently long to treat conservatively, and the more difficulty in maintaining the patient out of shock the sooner surgery should be done; younger people may be temporized with longer but they bleed to death, too. Recurrent hemorrhage, whether hours or days after the initial episode, is an absolute indication for immediate operation. Intervention may be necessary in a patient who bleeds slowly over a period of several days. The most urgent indication for emergency operation is continued severe bleeding.

If laparotomy is chosen, and on exploration, the bowel is filled with blood and no obvious source of bleeding is found below the first part of the duodenum, gastrectomy appears advisable, even when no ulcer is felt. In effect, subtotal gastrectomy seems to me the practical way to explore the lower end of the esophagus, stomach, first and second portions of the duodenum, unless a very wide opening is made in the stomach throughout its extent and across the pylorus into the duodenum. Cutting across the pylorus leads to serious interference with the motor mechanism of the stomach and oftentimes sequelae are more disagreeable than after gastrectomy. It must also be remembered that the operation is a heroic undertaking and that resection carries with it 80 per cent probability of getting the lesion.

During the course of performing gastrectomy the duodenal stump can be opened and the second portion of the duodenum visualized. In exploring a patient recently for bleeding, I found a healed ulcer in the first part of the duodenum after dividing it; on closer inspection I found the bleeder in the base of an ulcer in the second part of the duodenum. The lesion could not be felt from the outside, and would have been overlooked if the duodenum had not been opened widely.

The same thing can be done at the upper end of the stomach after it is cut across. The remaining gastric mucosa can be carefully examined.

The fallacy of doing less than subtotal gastrectomy for duodenal ulcer in the face of recent hemorrhage is illustrated by one patient in the Charity Hospital series who came in with recurrent small hemorrhages A simple gastroenterostomy was done which was followed by hemorrhage and death. The ulcer should be removed, if possible, the vessel controlled, and resection done.

The commonly accepted treatment of perforated duodenal ulcer is surgical closure of the opening and the very excellent results in 83 patients treated with simple

closure at Charity Hospital bear this out (Figure 3). Within recent years two ex-

PERFORATED ULCER

	Number of PATIENTS	DEATHS	MORTALITY RATE
TOTAL	105	12	11.4%
SURGERY	90	2	2.2%
Simple Closure	83	2	2.4%
Gastric Resection	7	0	0

Figure 3. These resections were carried out at the time of perforation.

tremes in management have been advocated.

Nonoperative methods of treating perforation embrace the principles of intravenous alimentation, continuous gastric suction, and antibiotics. Originally advocated by Wangensteen for the doubtful case, the poor risk patient, the localized perforation, and the late case with generalized peritonitis which has sealed off, it was revived in 1945 by Bedford-Turner to include all perforations.

The rationale of the method is that the ulcer has sealed off before surgery and manipulation breaks down the natural barriers. An added factor is that surgery may be undertaken for conditions such as coronary heart disease which mimic perforated ulcer. From the economic standpoint the man who has not been operated on will return to his work sooner than he who has. Against these arguments are that most frequently the ulcer is not sealed off, and contrarily, an erroneous diagnosis of perforated ulcer is more likely made in situations demanding immediate surgery, such as appendicitis.

Although a long time has elapsed since Von Haberer, in 1919, first recommended gastrectomy for the treatment of perforated ulcer, its more widespread use has only recently been accepted. Turner [9] recently reported that in 147 patients with duodenal perforations followed twenty years, 85 per cent had symptoms and one third required surgery. His conclusion from the study was that perforation signifies intractability and that subtotal gastric resection should be carried out, either at the time of perforation or within a short convalescent period. If resection is done, it should be used only in a patient seen soon after perforation whose general condition is good. If these limitations are observed the method should have as low a mortality rate as simple closure of the perforation, with better final results.

Most gastric surgeons commonly employ subtotal resection (See Figure 4). The

SURGICAL PROCEDURES

TYPE of GASTRIC RESECTION	NUMBER	DEATHS	MORTALITY RATE
BILROTH II (Gd)	187	10	5.4%
BILROTH I (GD) (VAGOTOMY IN 4)	8	1	12.5%
GASTROENTEROSTOMY (VAGOTOMY IN 3)	12	3	25.0%
EXPLORATORY CELIOTOMY	1	0	0
VAGOTOMY	1	0	0
CLOSURE PERFORATION	83	2	2.4%
TOTAL	292	16	5.5%

Figure 4

mortality rate is low, the postoperative morbidity minimal, and the hospital stay is less than two weeks.

Seventy to 85 per cent of the stomach must be removed, including all of the pyloric mucosa, to safeguard against recurrent ulceration. The great hazard in gastric resection is in obtaining secure duodenal stump closure. If ulcer formation is a secondary effect of excessive acid secretion and gastrectomy effects acid reduction, the ulcer does not have to be removed. The pyloric mucosa must be excised and if closure presents a problem, the Bancroft procedure [1] is a useful one (See Figure 5). Preferably, the ulcer should be gotten out.

Another safety maneuver in difficult closure is insertion of a catheter in the stump. The catheter is taken out in ten to fourteen days and the fistula will close.

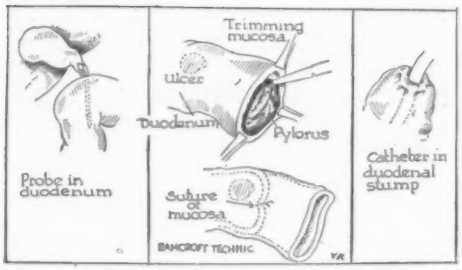

Figure 5. Safeguards in management of the duodenal stump.

A probe in the common duct, introduced from above or below, will help in protecting it from operative injury. No suitable method has been found to avert pancreatic damage and increasing incidence of surgical insult to the pancreas is being discovered. Many late digestive disturbances are based on pancreatic dysfunctions which have their beginning at the operating table.

Numerous technical variations in gastrojejunal anastomosis with gastrectomy designed to decrease the incidence of dumping syndrome include creation of a small stoma, a valve at the afferent side, antecolic anastomosis with afferent loop on the greater curvature, and additional peritoneal support of the gastric remnant.

Gastric resection with gastroduodenal anastomosis (Billroth I) is proposed by Harkins and others.[5] The objections to gastrojejunal anastomosis are the decreased pancreatic and biliary secretion resulting from impairment in the stimulating mechanism, imperfect mixing of food with these secretions because the gastric contents are shunted away, and increased motility of the bowel. Gastroduodenostomy cannot be done with a stenosed, inflamed, or fixed duodenum, a fixed stomach, or in lesions that require high resection along the greater curvature. Gastric retention is a frequent postoperative complication.

The only other competitor of gastric resection is vagotomy (See Figure 6). Alone, it has no place in the treatment of duodenal ulcer. Combined with gastroenterostomy it has a large number of advocates. It should not be performed as an emergency procedure for active hemorrhage. The Committee on Surgical Pro-

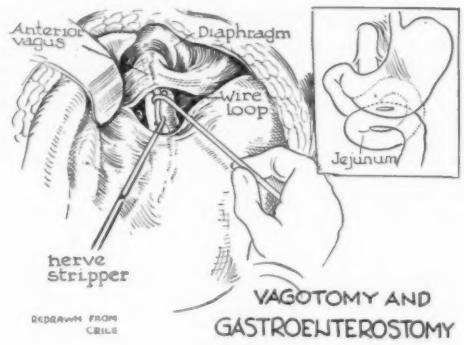

Figure 6

cedures of the American Gastroenterological Association[12] reported in November 1952, that a 70 or more per cent gastric resection (96.7 per cent good results) is a more satisfactory procedure for duodenal ulcer than gastroenterostomy and vagotomy (93.7 per cent good results).

To decrease the incidence of gastrojejunal ulceration which occurs in 3 to 8 per cent of patients undergoing vagotomy or resection, a combination of the two has been recommended. The hormonal and cephalic phases of gastric secretion are thereby abolished. Nothwithstanding the report of the Surgical Procedures Committee to the effect that vagotomy does not decrease the possibility of recurrence when a 70 per cent or more resection is done, Druckerman et al[2] have used the two together in 165 cases and no recurrent ulcers have been observed. Postoperative morbidity is increased slightly. The approach seems to have a great deal of merit and a larger experience may lead to its adoption by most surgeons.

The simple procedure of gastroenterostomy has its ups and downs, its past abuses, and its present limitations. Its usefulness in elderly patients with pyloric stenosis and low acidity cannot be denied. Care of the patient in the postoperative period may be more difficult than with gastrectomy because of the dilated stomach which poses more of a problem in

electrolyte control and prevention of regurgitation. One patient in the Charity Hospital series drowned in his own gastric juice after gastroenterostomy when the gastric suction was not working properly.

A combination of vagotomy and 50 per cent gastric resection, as advocated by Smithwick, probably has little more to offer than does vagotomy and gastroenterostomy. Wangensteen [10] has recently revived segmental resection of the stomach, and tubular resection, the latter method being a removal of all the greater curvature of the stomach from the esophagus to the duodenum, leaving the lesser curve as a small gastric tube in continuity. Moroney [6] (1951) published a series of successful cases in which he replaces the excised stomach with a detached portion of the transverse colon (Figure 7).

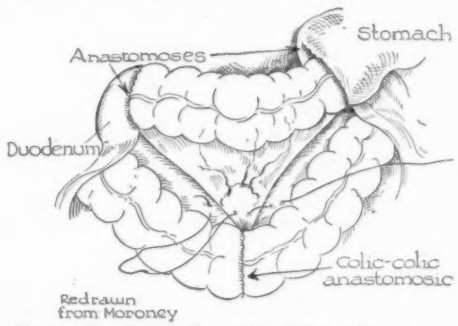

Figure 7. Stomach replaced by colon.

In spite of all the surgical procedures that have been proposed, recurrent ulceration will take place occasionally. Gray's hypothesis (See Figure 8) of a separate

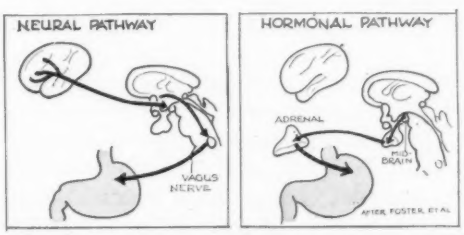

Figure 8. Hormonal mechanism as a possible etiologic factor in recurrent ulceration after gastrectomy or vagotomy [7]

hormonal factor acting directly on the gastric glands and mediated by the pituitary-adrenal axis gives insight as to why ulcer recurs. Dragstedt administered cortisone subcutaneously to dogs for periods up to a month which produced a sustained increase in gastric secretions. The stimulating action was noted in dogs with vagotomized and nonvagotomized stomach pouches and in animals with isolated stomach pouches in which the antrum had been removed.

Recent studies of electrolyte exchanges in the postgastrectomy dumping syndrome show that after ingestion of a meal, glucose is rapidly deposited in liver and muscle, potassium is shifted suddenly, and temporary extracellular potassium is shifted suddenly, and temporary extracellular potassium deficit exists. [4] At just what phase of the dumping syndrome potassium changes occur has not been definitely worked out and reports are in conflict as to the efficiency of potassium by mouth in treatment of the dumping syndrome. [8]

SUMMARY

Surgery in duodenal ulcer is reserved for its complications, hemorrhage, perforation and obstruction. Penetration is perforation and when a patient with ulcer develops continuous pain radiating through to the back, unrelieved by food and ulcer medication, he has perforated.

A person under 20 or over 60 having a complicated ulcer should have an early surgical appraisal.

Continued severe bleeding is an urgent indication for emergency operation. If no bleeding point is found, subtotal gastrectomy is favored.

Perforation of an ulcer signifies intractability and resection should be carried out.

A 70 or more per cent gastric resection accompanied by gastrojejunostomy is a more satisfactory procedure for duodenal ulcer than is gastroenterostomy and vagotomy, or resection with gastroduodenostomy, or any other procedures now being employed, with the possible exception of combined gastric resection and vagotomy. Combination of the two procedures which

effectively abolishes cephalic and antral phases of secretion has considerable promise.

The two common aftermaths of gastrectomy are dumping and recurrent ulceration. Abnormal shift of potassium and other electrolytes has been noted in the dumping syndrome. Gray's hypothesis of an adrenal hormone acting directly on the remaining gastric glands to increase acid secretion may account, at least in part, for gastrojejunal ulcer after gastrectomy.

Charts on 623 patients with duodenal ulcer admitted to Charity Hospital during 1951-1953 have been available for study. Forty patients died (6.4 per cent mortality). The mortality rate in 83 patients treated with simple surgical closure of a perforated ulcer was 2.2 per cent. The over-all mortality rate in bleeding duodenal ulcer was 9.2 per cent. Twenty per cent of those operated during active hemorrhage died. Subtotal gastric resection with gastrojejunostomy, including those who had emergency surgery, carried with it a 5.4 per cent mortality.

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THE DISADVANTAGES OF URETEROSIGMOIDOSTOMY *

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While much has been written about diversion of the urine into the bowel for a variety of defects and diseases of the bladder, relatively little has been said about the late complications and end results of this procedure, and its effect upon longevity. Most papers have dealt with technique; anyone who has read the monumental review of Hinman and Weyrauch (1937) knows that innumerable variations have been advocated; many more have been reported since their paper.

There is no doubt that ureterosigmoidostomy can be compatible with long survival. Thus Stevenson, in 1941, discovered a patient living forty-three years after ureteral transplantation for exstrophy of the bladder. He collected reports of 11 others surviving twenty to thirty years. Lower found that 6 of his patients had lived or were living more than twenty years after operation. Jacobs, discussing his personal experience with 138 ureterosigmoidostomies, mentions 6 who lived ten years or more. Hinman, Jr. has collected reports of the birth of 33 babies to 26 women with ureterosigmoidostomy, and has added 2 of his own.

Furthermore, the widespread application of transfusion and the effective use of antibiotics have greatly reduced surgical mortality. However, G. G. Smith has well said "but until the late results of ureteroenterostomy show a much lower

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incidence of renal failure, one hesitates to employ the method for a condition which is not in itself dangerous to life." Hinman remarked that it was only an occasional brilliant result that encouraged him to persist in his efforts to perfect the operation.

The reasons for these views are plain. The principle of the operation (the connection of the normally sterile upper urinary tract to the normally infected colon) is unphysiological, and will remain so until someone discovers a universal, nontoxic antibiotic to which bacterial resistance never develops.

The problem is not one of infection alone, since several other factors initiate and aggravate its effects. First, the ureters must be mobilized for some distance to permit their anastomosis to the bowel, thus damaging their extrinsic blood supply. While intrinsic vessels ordinarily prevent necrosis, the impaired vascularity may cause temporary edema and start the almost inevitable dilatation of the upper tract. The same process may reduce ureteral resistance to infection, and result in late stricture at the cut end of the ureter. This danger is presumably lessened but not entirely eliminated by slitting one side of the end of the ureter and anastomosing it directly to the bowel (Nesbit) so that there is no free, partly devascularized end protruding into the colon.

A second danger inherent in all methods, except Nesbit's and possibly Cordonnier's, is that of compression, at first by inflammatory edema and later by scarring, of the ureter by the wall of the colon. This hazard is doubtless reduced by preoperative preparation of the bowel with oral antibiotics, and by their parenteral use during convalescence. Baker has presented some experimental evidence suggesting that the postoperative administration of cortisone will minimize fibrosis in the incision in the colon, but this needs both experimental and clinical confirmation.

A third danger is that of reflux of feces into the ureter. This is fostered by the anastomoses of Nesbit and of Cordonnier, since both depend upon ureteral peristalsis to prevent regurgitation. Whisenand and Moore have reported a case in which Nesbit's operation in a patient with bilateral nephrostomies was followed by the escape of gas and feces from the renal catheters until they were removed, after which recovery occurred! Several observers (Parks, Mathis, Turner and Saint) have seen gas in the kidneys and ureters in roentgenograms made after such operations. Woodruff, Cooper and Leadbetter, Riba, and Weyrauch and Young have all shown by animal experiments that the submuscular tunnel of Coffey greatly lessens the danger of reflux. The prevention of regurgitation was Maydl's object in anastomosing the whole vesical trigone to the sigmoid, but Baker and Miller have shown, in the dog at least, that the intracolonic pressure finally overcomes even the normal ureterovesical valve when the defunctionalized colon is anastomosed to the bladder.

Mathisen has recently presented an interesting variant of ureterosigmoidostomy intended to prevent the entrance of feces into the ureters. He wraps the ureter in a quadrangular pedunculated flap of intestinal wall which is made to protrude into the lumen of the sigmoid. The objectives are to support the protruding ureter, and to permit its compression both by colonic peristalsis and by intestinal pressure. He was "unable to demonstrate reflux" in 18 clinical cases after this operation, but his longest follow-up was one year. Several dogs in which we did this operation developed strictures at the ends of the ureters in a very short time.

The route of infection of the kidneys after ureterosigmoidostomy is obvious. In addition to the lumen of the ureter, organisms can follow the periureteral lymphatics, since Hinman, Sr. found that the dog's kidney became infected when the unopened ureter was buried in a trough in the wall of the colon.

Statistics show the frequency and seriousness of obstruction and infection aft-

er ureterointestinal anastomosis. Harvard and Thompson blamed pyelonephritis for 50 percent of the surgical and 67.5 percent of the late deaths in their series of 98 cases followed for five to thirty-five years after ureterosigmoidostomy for exstrophy. Smith investigated 54 survivors of ureterosigmoidostomy. Nine died of "renal failure" within two years; 4 more required cutaneous ureterostomy for infected hydronephrosis, and 3 others had poor renal function at the time of investigation. Thus 16 of 54 patients (29.6 percent) had severe renal damage within a few years. Twenty-one of Cordonnier and Lage's 54 patients (38.9 percent) showed evidence of pyelonephritis within thirty months of operation. Lapides had never seen at autopsy a kidney which had been connected to the bowel and remained free of infection either in dog or in man.

In 258 patients subjected to urography six months or more after operation (53 from the University Hospital and 205 from the literature) only 31 percent had normal appearing kidneys. Since most of the urograms were made within a year of operation, the long term outlook is gloomy indeed.

The dangers of hydronephrosis and pyelonephritis after ureterocolic anastomosis are not limited to uremia and sepsis, but involve the complication of hyperchloremic acidosis. While Jewett and later Ferris and Odel have amply demonstrated that this results from absorption of urinary constituents from the colon, Lapides has proved, by ingenious clinical experiments, that renal injury aggravates the condition because it impairs excretion of absorbed chlorides and other acid radicles. This was foreshadowed by Ferris and Odel's observation that unilateral nephrostomy could relieve hyperchloremic acidosis even though the other kidney continued to secrete its urine into the colon. Our own studies have shown that hyperchloremic acidosis occurs three times as often and causes symptoms ten times as frequently when the kidneys are damaged as when they appear normal, and that it cannot be prevented or relieved by shortening the loop of bowel receiving the urine.

All this does not mean that ureterosigmoidostomy should be abandoned, but rather that it should be used only when there is no acceptable alternative, and that efforts to protect the kidneys afterward should be continued. What measures do we have at our disposal now, pending the development of a universal, nontoxic, totipotent, never-failing antibiotic to which bacterial resistance cannot develop?

POSSIBLE SOLUTIONS TO THE PROBLEM

If diversion of the urine is required in the presence of bilateral hydronephrosis or of nitrogen retention, cutaneous ureterostomy with all its disadvantages, is preferable to ureterosigmoidostomy because of the danger of hyperchloremic acidosis with the latter. In good risks one may consider Bricker's anastomosis of the ureters to a short, isolated loop of ileum with an external stoma to which a Rutzen or similar bag can be fitted.

When the ureters are to be anastomosed to the colon, the latter should be prepared with care. Initial catharsis is followed by the "preoperative colon diet" of Bargen. Neomycin and polymyxin B (aerosporin) are given orally for three days before operation, and colon flushes are used on the last evening. Since feces cultured at operation after this regimen may be sterile, its use should promote smooth healing of the anastomosis.

Poth has recently reported sterile fecal cultures four days after a twenty-four hour regimen consisting of catharsis followed by oral neomycin and sulfathalidine; this sounds like a superior method.

Certainly, the anastomosis should be of the terminolateral type favored by Nesbit in order to increase the circumference of the stoma, to leave no bare, devascularized ureter protruding into the bowel, and thus to discourage stricture formation. This should be supplemented by the construction of a periureteral tunnel composed of intestinal muscle and mucosa in order to minimize reflux.

Healing should be favored by the use of potent antibiotics parenterally during convalescence, and Baker's suggestion that cortisone be used should be investigated more extensively in the dog, although the literature concerning the effect of cortisone upon the healing of wounds casts doubt on the validity of Baker's assumption.

Terminal colostomy with closure of the proximal end of the distal loop should be given a thorough trial because, with modern antibiotics, the colon which receives the urine can certainly be made and kept clean enough to offer considerable protection to the kidneys which certainly need every possible help. While this measure was suggested by Krönig in 1907 and even earlier by Mauclaire, it has never had a fair trial for obvious reasons. Vest has recently reported securing sterile urine from the bowel nine months after a vesicosigmoidostomy of his own devising for exstrophy, a fact which should certainly encourage studies in this direction.

SUMMARY AND CONCLUSIONS

1. Ureteroenterostomy subjects the kidneys to great risk of serious damage from obstruction (stricture and reflux) and infection (reflux and ascent of periureteral lymphatics).

2. When serious renal damage follows this operation, hyperchloremic acidosis becomes a problem.

3. Cutaneous ureterostomy is preferred when renal damage antedates operation.

4. Ureteral anastomosis to the bowel should be used only when the underlying disorder threatens life or makes it intolerable, and no satisfactory alterntive is available.

5. Means at hand to minimize postoperative renal damage, at least theoretically, include: Careful preparation of the bowel; the use of an operation designed to prevent stenosis of the anastomosis as well as reflux; the administration of antibiotics and possibly of cortisone during healing; and the complete diversion of the fecal stream above the anastomosis.

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EXPERIMENTAL VISCERAL DISTURBANCES OF CEREBRAL ORIGIN IN RELATION TO MECHANISMS OF CONVULSIVE THERAPY

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We are still remote from a satisfying explanation of the therapeutic mechanisms of the convulsive procedures. Biophysical and biochemical changes in the brain accompany and follow the reaction and a part, at any rate, of the response is an increased excitability of the cerebral neurone. If we examine, post mortem, the brains of patients who have undergone electric or other forms of shock, we may discern evidence of destructive changes in the nerve cells. Are these changes due to the treatment and, if so, is neuronal destruction an essential element in the therapeutic effect? Does the treatment in some way establish more favorable metabolic patterns in the brain? To what extent may enzymatic and hormonal factors be altered? These are questions that press for answers.

We may expect at least partial answers to these questions. There is, however, a profound sense in which we must confess that the precise relationship between mental and emotional experiences on the one hand and neurophysiological action and neuropathological processes on the other continues to elude us. The mechanisms of the brain and the mechanisms of the mind are not yet conformable to a single pattern, although research on the prefrontal areas, for example, during the past twenty years has done much to bridge, for some, the gap between psychological and neurological functions. For others, like Sherrington[6, 7] the mind remains forever something apart, invisible, intangible, supreme, naked, and untouched. To Sherrington, the liaison between the psychical and the physical was an unsolved mystery.

"What right," he asked, "have we to conjoin mental experience with physiological" . . . To many of us a mere juxtaposition of the two sets of happenings proclaims their disparity. On the one side changing electrical potentials with thermal and chemical action making a physiological entity held together by energy relations; on the other, a suite of mental experience, an activity no doubt, but in what if any relation to energy? As for me, what little I know of the how of the one, does not, speaking personally, even begin to help me toward the how of the other. The two, for all I can do, seem to remain disparate and disconnected".[6] Sherrington has presented, with sensitive insight, the essential elements of the mystery. Although our ignorance may sometimes bid us despair, we may be confident that man's curiosity will lead him to seek and formulate new and more comprehensive syntheses in which he will envisage his psychological and physiological nature welded into a more perfect unity.

The object of my paper this morning is to share with you certain experimental evidence suggesting that the effects of therapeutic shock are not limited to action upon the brain alone but that the stimulus to the brain activates cortical autonomic centers by which widespread vasomotor (and other autonomic) changes are produced throughout the body. These changes must be taken into account in our thinking of the therapeutic mechanisms of shock procedures.

My own interest in the role of the cerebral cortex in the integration and coordination of autonomic as well as somatic functions was captured by reading, several years ago, the writings of Hughlings Jackson. This farsighted pioneer in neu-

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rological thought had, by 1869, concluded that the central nervous system, including its highest heirarchial levels, was concerned with total function—autonomic as well as somatic. Hughlings Jackson urged that the very highest processes (the substrata of consciousness) are only the most multifold and complex of sensorimotor processes and that they represent or re-represent *all* lower nervous centers and thus the organism *as a whole*. He explicitly affirmed that one must add to the constitution of the units of cerebrum nerve fibers to the heart, blood vessels, and viscera "or rather probably to the regions of the sympathetic system, from which these parts are supplied . . . The highest nervous processes are potentially the whole organism". [5]

Hughlings Jackson's holistic concept of brain function has a particularly modern appeal and there exists now an imposing and convincing body of experimental evidence for cortical representation of autonomic activity. Our own earlier contributions, reported in 1936, [2] constituted first an exploration of electrically excitable cortical areas in cats, monkeys, and the chimpanzee in search of those regions yielding vasomotor responses. We were able regularly to produce, in anesthetized, curarized animals, transient elevations of blood pressure, dilatation of the pupils, and retraction of the nictitating membranes by controlled, localized electrical stimulation of cortical loci in the frontal lobe. As shown in Fig. 1, the regions of lowest threshold for pressor responses in the cat include the anterior sigmoid gyrus, gyrus proreus, and the mesial portion of the orbital surface of the frontal lobe as far posteriorly as the lamina terminalis. Stimulation of the lateral aspects of the orbital surface, the cingulate gyrus, the lateral and posterior sigmoid gyri, and also the rostral portion of the pyriform lobe yields similar pressor responses but with higher threshold, lower intensity, and longer latency. As this figure also shows, there is a widespread area from which falls of arterial blood pressure

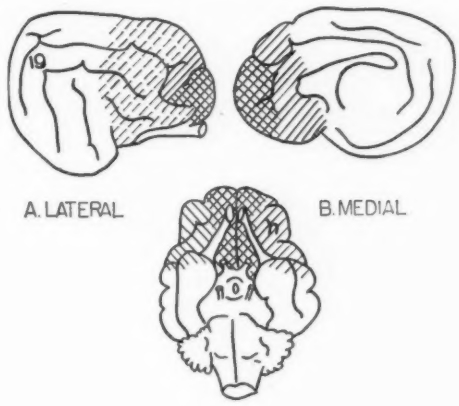

Fig. 1. A, lateral, B, medial and C, basilar surfaces of the cat's cerebral cortex showing areas yielding pressor and depressor responses to electrical stimulation. Crosshatched areas—rise of blood pressure, lowest threshold. Solid lines—rise of blood pressure, highest threshold. Broken lines —fall of blood pressure.

are obtained. This area includes the convexity of the hemisphere posterior to the sigmoid gyri and extending about halfway to the occipital pole or approximately as far back as the pseudosylvian fissure. Further posteriorly, no autonomic responses whatever can be elicited by localized electrical stimulation.

Figure 2 illustrates a rise of 60 mm. Hg in the mean arterial pressure with pupillary dilatation and retraction of the nictitating membranes from stimulation of a locus on left gyrus proreus of a cat. In Fig. 3 is seen a depression of 20 to 30 mm. Hg mean blood pressure following stimulation of a locus on left gyrus ectosylvius in the electrical depressor area of the cat. In all of the animals in these experiments, somatic muscular movements were totally abolished by d-tubocurarine. Ether anesthesia was used. Local anesthesia of the responsive cortical loci with dibucaine was found to eliminate the autonomic effects so the responses are the result of excitation of cortical elements and are not due to current spread to lower centers.

In the monkey, blood pressure changes are obtained from the motor and premo-

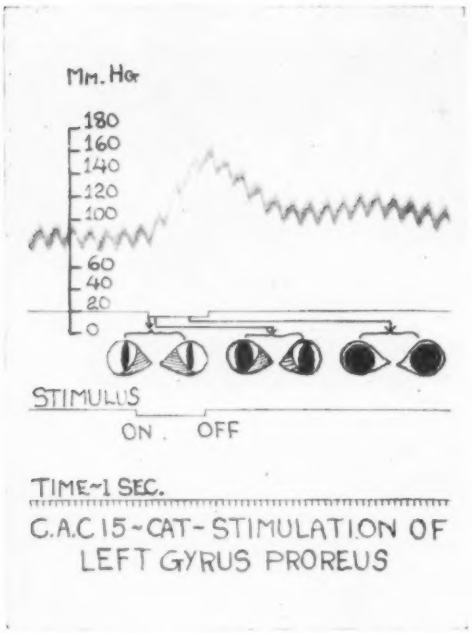

Fig. 2. Transient elevation of arterial blood pressure evoked by electrical stimulation of a locus on left gyrus proreus of a cat. Pupillary dilatation and retraction of the nictitating membranes are diagrammatically illustrated. (From Hoff, et al. [4])

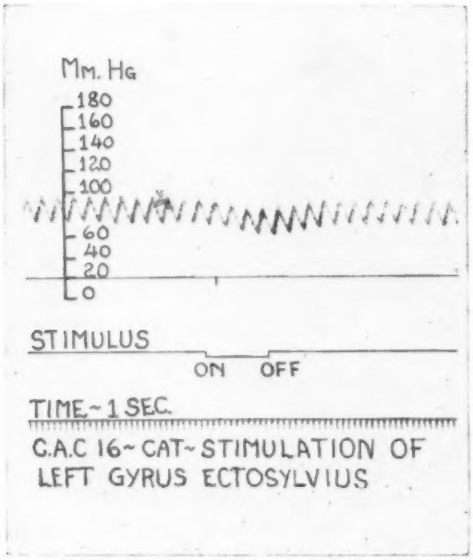

Fig. 3. Transient fall of arterial blood pressure evoked by electrical stimulation of a locus on left gyrus ectosylvius (in the electrical depressor area, Fig. 1A) of a cat.

tor areas (Brodmann's Areas 4 and 6) and from scattered loci rostrally on the convexity of the frontal lobe. Responsive areas on the orbtal and medial surfaces of the frontal lobe are likewise distinguished and the excitable region for vasomotor responses also includes the rostral tip of the temporal lobe. In the cat and the monkey, therefore, the cortical autonomic area overlaps the somatic sensorimotor region, and in 1939, we were able to report pressor responses from the motor cortex of human patients.[3] The cortical regional differentiation between pressor and depressor responses, seen in the cat, has not been demonstrated in the monkey, chimpanzee, or man.

In an analysis of the cortically-evoked pressor response, we found, in 1937,[1] that there is an associated widespread visceral vasoconstriction involving the kidneys, alimentary tract, and liver, and even in some cases, the heart and the brain itself. Figure 4 illustrates a typical effect of lo-

Fig. 4. Simultaneous dilatation of the forelimbs, reduction of renal volume, and elevation of arterial blood pressure from stimulation of the right lateral sigmoid gyrus of the cat's cortex. LL = left foreleg; † = increased volume; RL = right foreleg; ‡ = increased volume; RV = renal volume; BP = arterial blood pressure; Left Scale = mm. Hg; Right Scale = magnitude of volume change in cc. (From Green and Hoff [1])

calized electrical stimulation of a point on the lateral sigmoid gyrus of the cat. Here we observe a rise of arterial pressure with a synchronous reduction in kidney volume and a dilatation of both forelimbs.

These experiments, which have only been briefly touched upon here, offer evidence that there are neuronal elements within the cortex, excitation of which initiates a redistribution of blood, decreasing the supply to certain viscera (the kidneys, alimentary tract, liver, etc.) and increasing the supply to those parts (e. g the limbs) which would be made active by excitation of somatic efferent pathways from the same cortical areas. The

cortical autonomic mechanism thus serves with the cortical somatic mechanism to integrate a total response, with appropriate motor and vascular components.

The vasoconstriction in the kidney was not only inferred by reduction of renal volume but also determined by observation of blanching of the kidneys seen in white light or "black-out" of the kidneys under ultraviolet light with fluorescein in the circulation or by other methods such as India ink injections.[4] Figure 5 shows

Fig. 5. Cut surface of the left kidney of a control cat in which all of the surgical procedures were carried out without stimulation of the brain. The organ is well filled with blood.

the normal appearance of the cut surface of the left kidney of a control cat revealing that the organ is well filled with blood. In contrast, we see in Fig. 6 the kidneys of a cat subjected to electrical stimulation of a locus on the anterior sigmoid gyrus twenty times during an acute experiment lasting two hours. There is striking cortical and medullary ischemia with the juxtamedullary region still filled with blood.

During the past three years, we have imposed electric shock type stimulation upon the brain of the cat in acute and chronic experiments, using bilaterally applied percutaneous electrodes without anesthesia and with or without motor paralysis by d-tubocurarine.[4] For these experiments, the Offner Brief Stimulus Therapy Apparatus (BST) was employed with a resistance of 1,000 to 1,500 ohms

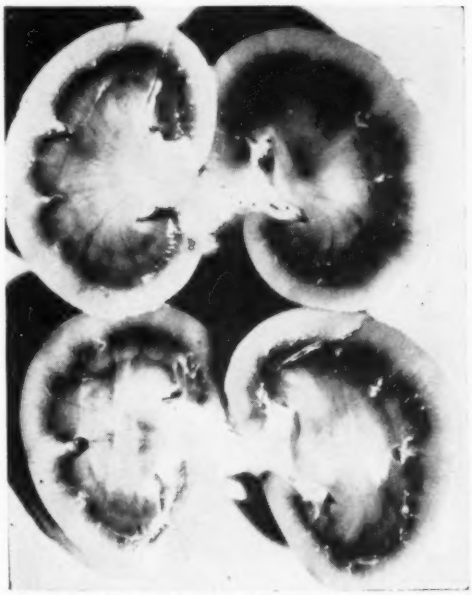

Fig. 6. Cut surfaces of the kidneys of a cat subjected to localized electrical stimulation of a point on the anterior sigmoid gyrus 20 times during an acute experiment lasting 2 hours. In contrast with the control kidney (Fig. 5) the cortex and medulla are ischemic. The juxtamedullary region is well filled with blood.

between the electrodes. The optimum pulse duration was 0.5 millisecond; the stimulus duration was 2 seconds; the peak current varied between 150 and 300 milliamperes and the average current between 10 and 18 milliamperes. With the electrodes in alignment with the cortical depressor region, falls of blood pressure were regularly evoked; when the electrodes spanned the cortical pressor area, rises of pressure were always produced.

Following multiple BST stimulations in acute experiments lasting two to three hours, there was fatty degeneration in the tubules of the kidneys. Figure 7 shows this fatty change in the renal cortex of a cat that received 19 BST stimulations in an experiment lasting three hours. Note in this hematoxylin and eosin stained section large fat globules in the convoluted tubules. In all the control kidneys little or no stained fat is revealed in Sudan IV sections.

Cats receiving 10 to 20 BST stimula-

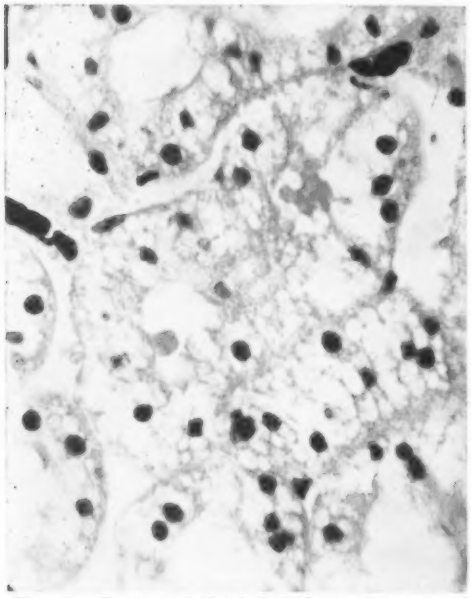

Fig. 7. Cortex of the left kidney of a cat subjected to 19 BST stimulations of the brain in an acute experiment. There is fatty degeneration of the tubule cells with pyknosis and fragmentation of the nuclei. Hematoxylin and eosin stain. (From Hoff, et al. [4])

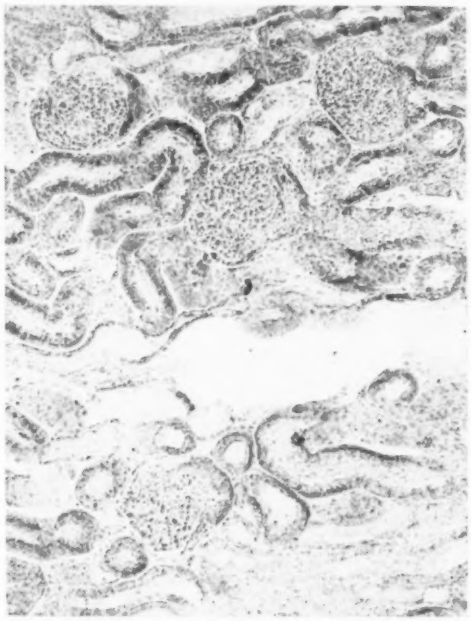

Fig. 8. Cortex of the left kidney of a cat subjected to 10 BST stimulations of the brain 7 days before sacrifice. There is severe degeneration of the tubules. The glomerular tufts are swollen and the glomerular spaces are filled with exudate and cellular debris. Sudan IV stain.

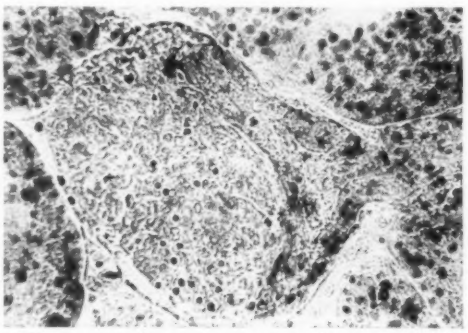

Fig. 9. A single glomerulus from the kidney illustrated in Fig. 8. (From a cat subjected to 10 BST stimulations of the brain 7 days before sacrifice.) Large fat globules are present in adjoining tubule cells; the glomerular swelling and filling of the capsular space are clearly seen. Sudan IV stain.

tions each week for one to six weeks exhibited swelling and severe fatty degeneration of the cells of the convoluted tubules with some nuclear changes and shrinkage of the cell-bodies. The glomerular tufts were swollen and the capsular spaces were filled with exudate and cellular debris. There was also an increase in the number of cells in the glomerular capsule and the juxtacapsular region of the convoluted tubule. There were fatty, hyaline, and granular casts in the tubules. We see in Fig. 8 severe fatty degeneration of renal tubules, swelling of the glomeruli, and filling of the capsular spaces in the kidney of a cat given 10 BST stimulations of the brain seven days before sacrifice. Figure 9 is an enlarged microphotograph of a single glomerulus in the same animal and shows in detail the glomerular swelling, and the filling of the capsular space. These changes were widespread throughout the renal cortex and involved nearly all of the glomeruli and tubules observed. In a given kidney the vessels of some glomerular tufts were partially obliterated, others completely. (See Fig. 10).

In short, we have in the BST stimulated animal a picture of nephrosis based in all probability upon repeated arteriolar spasm in the kidney. This leads to anoxia

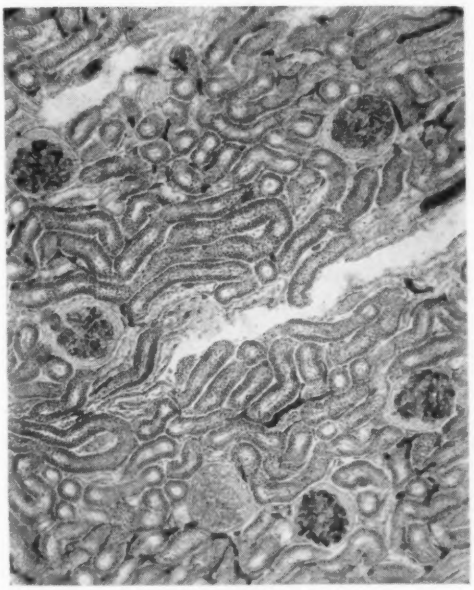

Fig. 10. Cortex of the left kidney of a cat subjected to 50 BST stimulations over a period of 30 days with sacrifice 9 days after the last stimulation. Intracarotid injection of India ink just before sacrifice. Hematoxylin and eosin stain. Glomerular spaces are filled with exudate and cells, and the vessels of the glomerular tufts are partially, and in one instance completely, uninjected with ink. Hoff, et al. [4])

of the glomeruli and tubules with degeneration, and with exudation due to permeability changes. No enduring hypertension has been seen and these changes are probably for the most part reversible. It may be that with a background of infection or other pre-existing complications, irreversible renal failure may result.

We have said that other viscera participate in the vasospasm produced by brain stimulation. Figure 11 shows at the right the stomach of a control cat in which the arterial system was injected just before sacrifice. Note that the organ is well filled with ink. Contrast this with the vasoconstriction and therefore failure to take up the ink in the stomach of a cat that had received 4 BST brain stimulations in an acute experiment. (Left-hand side, Fig. 11).

The vascoconstriction may involve even

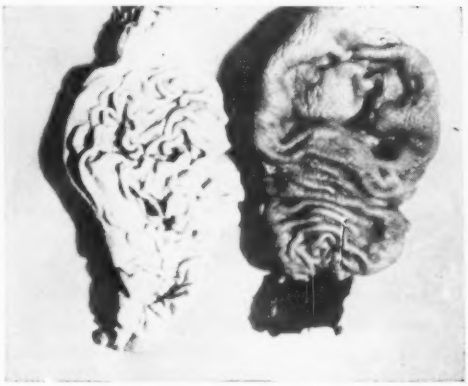

Fig. 11. Right—mucosal surface of the stomach of a control cat in which the arterial system was injected with 50 cc. of 50 per cent India ink just before sacrifice. The organ has freely taken up the injection mass. Left—mucosal surface of the stomach of a cat subjected to 4 BST stimulations of the brain in an acute experiment. India ink injection as in the control. Note failure to take up the injection mass.

the brain itself. In Fig. 12 we see on the left (Experiment 68) a brain from a control cat that has freely taken up the arterial India ink injection mass. Note next (Experiment 64) the vasoconstriction in the brain of a cat in which a locus on the

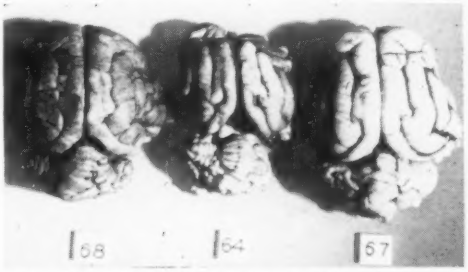

Fig. 12. Left ((Experiment 68)—Brain of a control cat; arterial injection of India ink as in Fig. 11. Middle (Experiment 64)—Brain of a cat that had received 7 electric stimulations of a locus on the left anterior sigmoid gyrus in an experiment lasting 3½ hours; arterial injection of India ink as above. Right (Experiment 67)—Control cat; no India ink injection. Contrast the vasoconstriction in Experiment 64 with the heavier injection in the control (Experiment 68).

left anterior sigmoid gyrus was stimulated seven times in an experiment lasting three and one-half hours. A noninjected control brain is seen at the right (Exper-

iment 67). The depth of the vasospasm in Experiment 64 may be judged in Fig. 13 which shows the same three brains in

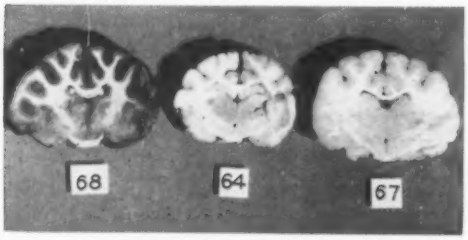

Fig. 13. Cross-sections of the three brains illustrated in Fig. 12. Arterial India ink injection as above. Contrast the vasoconstriction in the brain of the stimulated animal (Experiment 64) with the free injection of the brain in control (Experiment 68).

frontal section in the same order as in Fig. 12.

The lungs and spleen in all the brain stimulation experiments reported here consistently exhibited a plethora in contrast to the visceral vasoconstriction elsewhere. These organs were congested with blood and the lungs showed considerable hemorrhage. We have recently found in cats given repeated daily convulsive doses of metrazol over a period of seven weeks that there is a reactive medial hypertrophy of the pulmonary arteries. So although a cerebrogenic systemic high blood pressure has not been produced, it appears that in the course of these repeated experimental metrazol convulsions, pulmonary hypertension has supervened.

In conclusion, these experiments point to the existence of a physiological regulatory mechanism within the cerebral cortex whereby vasomotor changes appropriate to total body responses are effected. Typically, stimulation of the pressor area of the cerebral cortex initiates a redistribution of blood from certain viscera into active skeletal muscles at a raised level of arterial tension. Electrical stimulation of the cortex or the brain as a whole may lead to visceral vasospasm with resulting anoxic changes. Since these changes are widespread, probably involving the endocrine glands as well as the various organs discussed, it seems likely that the therapeutic effects of electric shock do not depend solely upon the action of electric current on the brain but involve also general metabolic alterations. Similar reasoning may be applied to other forms of convulsive therapy.

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TWO SIMPLE OFFICE TECHNIQUES IN PEDIATRIC PRACTICE

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SARAH MARK BRAUD, M. D.
THIBODAUX

1. A SIMPLE METHOD FOR IMMOBILIZATION OF CLAVICULAR FRACTURES IN INFANTS

During the newborn period, fractures of the clavicle are more frequent than all other fractures combined. These fractures nearly always occur in the lateral half of the clavicle. The site is usually at the junction of the lateral and middle third of the shaft. During passage of the infant through the birth canal, the clavicle is broken by excessive pressure of the anterior shoulder against the maternal symphysis pubis. In breech presentation, direct traction of the obstetrician's fingers in the attempt to express the shoulders is also responsible for clavicular injuries.

In addition to the above mentioned neonatal clavicular fractures, one frequently

encounters this condition in the latter part of the first year and early second year of life, in the so-called "toddlers." This injury usually occurs as a result of a fall on the outstretched hand, elbow, or shoulder, this being pushed violently against the chest.

The chief objectives in the treatment of the above conditions should be to make the infant as comfortable as possible with as little restriction of activity as is necessary, and yet obtain the maximum in a satisfactory functional and cosmetic result.

The following is a simple technique that I have used quite successfully for about two years in my office practice. Its chief advantage lies in economy and ease in availability of materials; also, in that the following dressing can be removed and reapplied by the parent in the home. This will permit bathing of this area of the infant.

Method: Only three materials are needed:

1. A large size diaper.
2. Two large safety pins.
3. Adhesive tape.

Step No. 1.—First a measuring tape is passed around the shoulders in the manner of a posterior figure-of-8, in order that the approximate length of the dressing to be used may be determined.

Step No. 2.—Next the diaper is opened to its full square area.

Step No. 3.—The diaper is now folded in a triangle.

Step No. 4.—By continuous folding in a 2 inch band, the diaper is folded to an elongated dressing.

Step No. 5.—The predetermined measurement of Step 1 is now applied to the dressing, and both ends are folded medially to accommodate this measurement.

Step No. 6.—The ends are taped by circular application of adhesive tape and also the center where the loose ends of the triangle are present.

Step No. 7.—Now the folded diaper band is applied as a clavical posterior figure-of-8 dressing and secured in place by the two safety pins.

No paddings are necessary, and the application of this dressing for about two weeks will suffice to permit adequate union. It is obvious that this method has its limitations in those cases with any marked degree of displacement.

2. SIMPLE TECHNIQUE FOR TREATMENT OF GRANULOMA OF THE UMBILICUS

In the vast majority of normal infants the umbilical cord will dry and divide by the sixth to the eighth day of life. Some slight degree of infection, usually saprophytic organisms, almost invariably occurs in the wound after the shedding of the cord. Normally thin skin and scar tissue form in the wound and healing is complete in two weeks. Some wounds remain open and do not heal for weeks. The most common cause for delayed healing is umbilical granuloma. This is a nubbin of exuberant "proud flesh" that extends upward from the base of the wound and prevents epithelialization. This tissue is identified as soft, vascular and granular, dull red or pink in color, and may have a seropurulent secretion.

Classically the treatment as described in most textbooks, has been by cauterization with silver nitrate sticks and the application of various powders and dressings. This usually takes weeks for healing, and occasional bleeding, purulent discharge, and even secondary cellulitis are sources of great concern to both parents and the attending physician.

The following is a simple technique that has been used with great success by me.

When the true character of the pathology has been ascertained to be a granuloma, the wound is cleaned with alcohol on a cotton applicator. An office assistant places superior and inferior traction with the fingers at the umbilical margins. This causes the base of the wound to flatten and protrude outward. Now a long length of No. 40 white cotton sterile ligature is placed around the granuloma and worked as close as possible to the base of the wound by means of a hemostat. This ligature is tied as tightly as possible, then ends are cut about 2½ inches long and allowed to lie out of the wound. Next the proud flesh

is cauterized in the classical manner with a silver nitrate stick, and alcohol is once again applied in wound with cotton tip swab. The traction is released and the granuloma falls back into the wound with the cotton-tie ends sticking out. No dressing is applied. The parent is instructed in the method of twice daily cleansing of the wound with a cotton tipped applicator. Eight grams of a liquid ointment * that contains 4000 units of polymyxin B sulfate and 200 units of bacitracin are prescribed with instructions to instill 3 to 4 drops twice daily in the umbilical wound.

Because the blood supply to the granuloma has been interrupted, in the majority of cases it will now separate and leave a clean base in about five to seven days. Only on one occasion has it been necessary to apply a second ligature; however, in rare cases it may be necessary to cauterize the proud flesh for the second time.

SUMMARY

Two simple office procedures have been described.

1. A diaper method for posterior figure-of-8 immobilization of clavicular fractures.

2. A cotton ligature method for dealing with granuloma of the umbilicus.

* Polycin liquid, (Pittman-Moore Co.)

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RETROLENTAL FIBROPLASIA;

AN OPHTHALMIC-PEDIATRIC-OBSTETRIC PROBLEM *

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GEORGE S. ELLIS, M. D.
TIMOTEO JIMENEZ, M. D.

NEW ORLEANS

Retrolental fibroplasia, a disease of the retina which destroys the vision of premature babies, was an unknown and unnamed clinical entity until 1942, when Terry [1] described the first case. Up to 1945, less than 175 cases had been recorded. Today this disease is the commonest cause of blindness in infants and young children.

Retrolental fibroplasia is no longer a matter of concern only to the ophthalmologist. It has, properly, become a matter of grave concern to pediatricians, obstetricians, and, most recently, epidemiologists. The pathologic process has been correctly described, but not very much else has been established, although the problem has occupied the attention of the best ophthalmic talent in the country for many years, aided, within the last few years, by other specialists, particularly pediatricians. Except that it is a disease of prematurity its etiology remains obscure. Furthermore, while we know how to diagnose it, we do not know how to prevent it or how to cure it.

The prognosis, unfortunately, is perfectly clear. Some cases regress spontaneously, but when once retrolental fibroplasia is fully developed, the end-result is total or almost total loss of vision. King's [2] report of 210 cases from the Massachusetts Eye and Ear Infirmary contains the longest follow-up studies on record, from two to seven years. Seventy-one of the children were totally blind at the end of the periods of observation. Seventy had light perception and could see hand movements. Forty-six could identify and reach for objects. The others, 10 per cent of the total number, had vision of 20/200 or better in at least one eye, though it was open to doubt, King stated, whether even this amount of sight could be retained.

Retrolental fibroplasia, however, is a disease which is so interesting and which, because of its enormously increased and increasing incidence, is of such importance, that it is our duty to take stock, at intervals, of the information available about it, even though what we know positively is discouragingly little.

Some of the titles in the literature make that fact clear. A recent editorial in the *Journal of the American Medical Association* [3] entitled it "an unsolved problem."

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This is, incidentally, the second editorial on the subject in this journal within a two and a half year period. Vail [4] called his article "The puzzle of retrolental fibroplasia." Speert and his associates [5] sum up most of what we know about the etiology in their subtitle, "A hazard of premature births." Locke and Reese [6] indicate the present status of our knowledge of the etiology in their subtitle, "The negative role of light, mydriatics and the ophthalmoscopic examination." They might have added the negative role of practically every other cause that has been investigated.

Zacharias' [7] excellent survey, published in 1952, contains 165 references to the periodical literature. I doubt that the listing is all-inclusive, but certainly this bibliography represents the most important articles on the subject which have appeared in the world literature from Terry's [1] original description in 1942 through the first months of 1952. The distribution of the papers (Fig. 1) is a significant

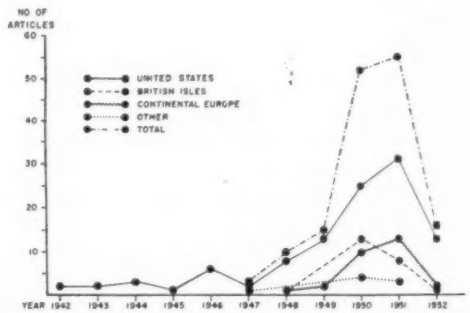

Fig. 1. Regional distribution of representative pericdical literature on retrolental fibroplasia. [7]

index of the geographic distribution of the disease, its increasing incidence, the increasing recognition of its importance, and the widening responsibility felt for it by ophthalmologists and nonophthalmologists alike.

The 59 articles listed from journals beyond the Continental United States include almost no 1952 publications. The largest number, 23, appeared in British journals. There are only single listings from five countries (Switzerland, Australia, Palestine, Cuba and Hawaii) and only two listings from several other countries. Most of the articles listed in the foreign literature were published in 1950 and 1951 (Fig. 2). There is only one for-

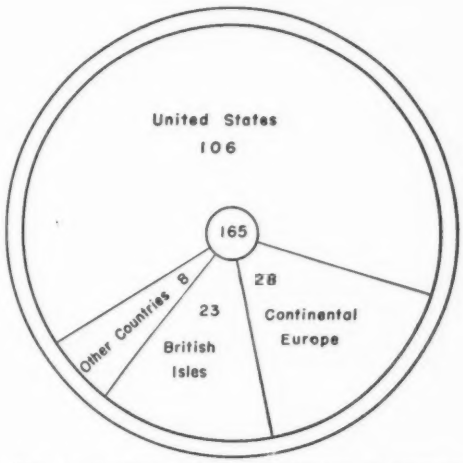

Fig. 2. Yearly regional distribution of representative periodical literature on retrolental fibroplasia. [7]

eign listing for 1947 and only two for 1948.

Of the 106 articles listed from the United States medical literature, 73 appeared in the specialty journals. There are two listings for 1942, and 56 of the 73 papers appeared after 1948. There are 15 listings from the pediatric journals, all after 1947 and all but four after 1949. The remaining 18 listings, most of them after 1948, include five from regional and five from state journals, three (exclusive of editorials) from the *Journal of the American Medical Association*, two from experimental journals, two from obstetric journals and one from a journal of public health. I do not know any more convincing demonstration of the increasing importance of this disease, or any better evidence of the widening interest in it, than this regional, chronologic, and specialty distribution of the literature on the subject.

In his first report on this disease Terry [1] called it a fibroblastic overgrowth of persistent vascular sheath behind the crystalline lens. As he continued his work

on it, the term retrolental fibroplasia was suggested to him by Harry K. Messenger,[8] and he accepted it as probably as satisfactory as any other that could be devised; the term, Terry pointed out, had not been used for any other condition and all the pathologic changes were apparently related to mesodermal overgrowth or persistence of embryonic mesodermal tissue.

When Krause[9] reported his first cases in 1946, he took the position that cerebral changes were part of the picture and that the disease should be called encephalo-ophthalmic dysplasia. It has since been pointed out that the cases he reported also included instances of retinal dysplasia. The retinopathy of prematurity, as suggested by Heath,[10] is a more reasonable term, if only because fibrous material occurs back of the lens in a number of other diseases. As often happens, however, although the concept of the etiology and pathology of the disease has changed, the term retrolental fibroplasia has continued in use. The nomenclature may not be precise, but it has become accepted by virtue of usage and it will probably continue to be used.

PATHOLOGIC PROCESS

Like many other apparently new diseases, retrolental fibroplasia seems to have existed for some time before it was recognized as such. Howard, in 1851, is usually credited with the first description, but Ryan[11] states that the first case was recorded by Travers in 1820. Whether all the cases recorded under other names were actually instances of what we now know as retrolental fibroplasia is perhaps open to doubt. For that matter, I am not so sure that all the cases reported since 1942 are actual instances of the disease.

At any rate, the credit for the first description of the disease belongs to Terry,[1] who in 1942 described a personally observed case and listed four others of which he had knowledge. The case which he observed, and the numerous cases recorded in the years immediately following his report, were characterized, in general, by the presence of an opaque, vascularized, retrolental membrane; a smaller than normal globe; a shallow anterior chamber; rather frequent anterior synechias; rather frequent detachment of the retina; and, if the ophthalmoscopic examination had been sufficiently thorough, the presence of elongated, dentate ciliary processes in the extreme periphery of the membrane behind the iris.

This is a perfectly correct picture of retrolental fibroplasia, but it is not the whole picture or the primary picture. A number of years were to pass before it was realized that it was the picture of the disease in its advanced stage, after the original process has been obscured by secondary changes. It was not until 1948 that Owens and Owens[12] from the Wilmer Institute reported the results of a study of 111 (of 214) premature infants who were followed for six months or more and 5 of whom developed retrolental fibroplasia. The study of these children from birth produced the following data:

In every instance the external appearance of the eyes was normal and examination of the fundus at first revealed no disease. The earliest detectable abnormality was a slight dilatation of the retinal arteries and veins. The dilatation gradually increased, particularly on the venous side; some of the veins were eventually three times their normal size. As the vessels increased in size, they also became tortuous. This was particularly true of the arteries, which often assumed the shape of hairpin curves.

The angiomatous dilatation and tortuosity of the vessels were soon followed by the appearance of one or more small, grayish-yellow elevations in the far periphery of the retina. Their identification was aided by the somewhat greater caliber of the vessels extending into the involved quadrant. These elevations, which were 1 to 3 diopters in height, were solid-looking and were slightly pigmented about the bases. Tortuous retinal vessels coursed over them, sometimes ascending the crest in a pattern like a crow's foot. As the elevations increased in size, the margins

of the disc became blurred, and generalized retinal edema developed. Other grayish-yellow areas were scattered throughout the fundus.

The next step in the pathologic process was the appearance of a grayish membrane, with numerous vessels coursing over it, which billowed forward in folds at the periphery of the retrolental space. Apparently it represented the anterior portion of the large masses of swollen retina. At this stage the fundus could be seen only hazily. Bands of tissue extended from the areas of detached retina into the vitreous, and between them a yellowish-red fundal reflex was visible.

In the final stage of the acute process a complete retrolental membrane was formed by gradual extension and fusion of the peripheral retinal folds. Broad ciliary processes extended onto the membrane at its periphery and numerous vessels were seen on its surface. At this stage the diameter of the cornea was much smaller than normal, the anterior chamber was shallow, posterior synechias formed, and the iris took on a grayish, atrophic appearance.

Secondary changes described by Owens and Owens were essentially those originally described by Terry.[1] They did not, however, appear in all cases. The primary process, in fact, did not go on to completion in all cases. The course of the disease might be interrupted at any stage, the pathologic changes remaining stationary or even regressing.

In 1951, after the observation of a larger series of cases, Owens and Owens reported with Friedenwald[13] that retrolental fibroplasia appears in three stages. The first stage is characterized by dilatation and tortuosity of the retinal vessels, with the development of new vessels and capillary varices. The second stage is characterized by the appearance of grayish retinal elevations, which may be localized or generalized, and small retinal hemorrhages. The third stage is characterized by the extension of newly formed vessels and supporting tissue from the retina into the vitreous, with detachment of the retina. The extent of the destruction in the final cicatricial stage depends upon the severity of the acute or active process and the amount of regression which occurs as activity subsides.

On the basis of a histologic study of 17 eyes secured from 10 patients, Friedenwald, Owens and Owens[13] reported the following observations:

The fundamental pathologic morphology in retrolental fibroplasia is proliferation of capillary endothelium with abnormal budding of capillaries in the nerve fiber layer of the retina. Surrounding the capillary proliferation are increased numbers of glial cells. Small areas of edema and hemorrhage may occur in the thickened nerve fiber layer. No inflammatory infiltrates are seen in the ocular tissues in these early stages. With further progression, small preretinal and vitreous hemorrhages occur. The newly formed capillaries break through the internal limiting membrane and extend over the surface of the retina and into the vitreous, accompanied by a delicate network of fibrous tissue suggestive of retinitis proliferans. Retinal detachment and disorganization follow. The retrolental membrane is formed by fusion of the detached retinal folds.

The studies of Owens and Owens[12] not only elucidated the pathologic process in retrolental fibroplasia, they had another important result. Terry had originally suggested that the cause of the disease was persistence of some part of the tunica vasculosa lentis system, the growth of embryonic tissue behind the crystalline lens, or the persistence of fibrillar structures of the vitreous humor. His idea was that premature birth occurs early enough for the hyaloid artery with its tunica vasculosa lentis and pupillary membrane to be patent. His reasoning was that since the hyaloid artery does not normally close until four to six weeks before term, prematurity might be an important factor not only in preventing its normal involution but also in producing an overgrowth

of supporting tissue. The studies from the Wilmer Institute eliminated this concept by the demonstration that the visible remnants of the hyaloid artery had disappeared before the onset of the disease. Similarly, these studies eliminated Reese and Payne's [14] hypothesis that the basic lesion in retrolental fibroplasia is persistence, in part or in toto, of the primary vitreous.

INCIDENCE

Perhaps the most extraordinary fact about retrolental fibroplasia is its increasing incidence in the United States. Only occasional and sporadic cases, which may or may not be instances of the disease, were recorded before 1942. Some cases were undoubtedly overlooked. One sees, occasionally, adolescents, or even adults, who have a lifetime history of impaired vision and whose examinations suggest that they may have had the disease, unrecognized, in childhood. The acute cases were certainly overlooked until 1949, when Owens and Owens [12] published their observations on this phase of the disease, and their inclusion in the series reported since that time explains some part of the recent increase.

It does not, however, explain all of it, or even the major part of it, nor does any other fact. Furthermore, no fact or theory explains the widely variable incidence in different parts of this country, or the variation between hospital and hospital in the same locality, or the far smaller incidence outside of the United States.

Retrolental fibroplasia was apparently originally confined to Boston, New York, and Chicago and areas immediately adjacent to these cities. Then it began to appear in other areas, and to appear in greater numbers, though in most irregular fashion. No cases were observed at the Wilmer Institute until 1945. [12] During the next three years the incidence was about stationary, between 15 and 20 per cent of all premature children. According to Krause, [15] the incidence in the University of Chicago Hospital in children weighing less than 1,500 gm. at birth rose from 11 per cent in 1945 to 36 per cent in 1946, 45 per cent in 1947, and 86 per cent in the first half of 1948. The incidence has fluctuated from year to year at the Boston Lying-In Hospital [16] and at the New York Eye Institute and the Babies Hospital. [5] The distribution of cases at the Babies Hospital,[17] during 1950 and 1951, is as remarkable as it is inexplicable: In spite of no measurable change in any phase of clinical management, including the use of oxygen, the disease developed in only 24 of the 230 infants born prematurely during 1950, against 56 in the 207 infants born in 1951.

As the literature indicates, retrolental fibroplasia appeared later in other countries than in the United States. [7] In most of them it is still an uncommon or actually rare disease. Furthermore, in Great Britain, where it is more frequent than it is in any country outside of the United States, it is appearing, according to Bembridge and his co-authors, [18] in the same curiously uneven and unpredictable fashion as in the United States. In Sweden, where a systematic search for cases was undertaken after the disease was first reported in 1948, the majority of cases were found in the populous districts and along the coast. [7]

The statement is frequently made that the disease is commoner in males, but as one examines series after series, the differences seem insignificant or nonexistent. In the 80 cases studied by Silverman and his associates, [17] for instance, at the Babies Hospital in New York, there were 39 boys and 41 girls. At the Boston City Lying-In Hospital [16] the sex distribution was approximately equal but in 1945, for some inexplicable reason, all 12 cases occurred in males. At the Providence Lying-In Hospital the male incidence was twice as high as the female. [16]

The racial incidence is hard to determine, one reason being that in many series no statement is made on this point. The reported series are chiefly made up of white babies, but one reason is that many of them consist chiefly of referred

private patients. Krause,[9] in 1946, stated that no cases had been observed at the Providence Hospital for Negroes. The figures from Charity Hospital of Louisiana at New Orleans are of dubious value since it is only relatively recently that the disease began to appear there. *

Social status plays no part in the incidence. The reported cases cover the financial range from wealth to indigence and the educational range from ignorance to the learned professions.

ETIOLOGIC CONSIDERATIONS

Prematurity.—The one indisputable etiologic fact in retrolental fibroplasia is that it is a disease of prematurity. It is only occasionally observed in full-term children. It is still true that the frequency is greater the more premature the children, but as the incidence is increasing, the disease seems to be extending into the more nearly mature groups. All the discussion goes back to what Terry[1] said in his first report in 1942, that some new factor has apparently arisen in extreme prematurity, though what the factor is remains to be demonstrated.

A number of observations have been made in respect to prematurity. There is universal agreement that the disease is most common in children of low birth weight. The highest incidence is in children weighing less than 4 pounds, and it is still fairly exceptional to find it in those weighing more than 4½ pounds. It was once true that the incidence was inversely proportional to the birth weight, but this situation is changing. Prior to 1943, according to Kinsey and Zacharias,[16] only 1 of 102 premature children at the Boston City Lying-In Hospital developed the disease in the weight group between 3 and 4 pounds. In the 1943-1947 period there were 33 cases in the 162 children in this group. Some observers believe that the smaller the child, the more likely it is to exhibit abnormalities on the first examination, and the more serious it is to find them at this time.

In their pioneer study of the disease in young infants Owens and Owens[12] found no cases in which the disease was present at birth and stated their belief that it is of postnatal origin. This is still the general belief, though occasional reliable observers have reported observing pathologic changes in newborn children.

In Owens and Owens' cases the first changes were usually observed between the second and fifth month. A number of other observers have reported the changes as early as the third week; I have myself seen them at this time. Guy and his associates,[19] who regard hazing of the vitreous as an early indication of the disease, have observed this change on the tenth day of life. The first changes have also been observed as late as the sixth month, but this is unusual. Guy's group formerly thought that if the ocular findings were normal within the first two weeks of life, the disease would not develop. Further observation convinced them that this point of view was not correct. Silverman and his associates[17] found, unexpectedly, that the disease had a later onset in smaller children and an earlier onset in larger children. They also found an inverse relationship between the birth weight and the period at which the first vascular signs of retrolental fibroplasia were noted. They could not establish a similar relationship between the age of clinical onset and the gestational age, though, with a single exception, clinical manifestations appeared in all of their 80 cases before the expected date of birth at full term.

Other Etiologic Factors.—Not a great deal of time need be spent discussing the possible etiologic factors in retrolental fibroplasia since, with few exceptions, most that have been considered have been promptly eliminated. The bilateral occur-

* The records of the New Orleans Charity Hospital show 16 proved cases of retrolental fibroplasia between 1949 and 1953, inclusive, and two additional possible cases. Two cases were observed in 1949, three in 1950, six in 1951, one in 1952, and four in 1953. There were 9 negro and 7 white children in the proved cases, and 7 males and 9 females. The period of gestation ranged from 5 to 8 months, with most of the cases concentrated in the sixth and seventh months. One child, not delivered in the hospital, was apparently full-term.

rence of the disease suggests a constitutional cause, but none has ever been identified. It would be tempting to postulate a familial or hereditary cause, but nothing in the disease warrants it and all the evidence is against it. The disease is frequent in twins and triplets in all reported series, but about all that can be read into that fact is that multiplicity is a common cause of premature birth. The occurrence of the disease in identical but not fraternal twins and triplets seems to lend support to the theory of genetic origin, but the disease also occurs in single twins and triplets, as well as in both children in nonidentical twinning.

The occurrence of other defects which might support the idea of a congenital origin is actually rather low. Certainly, they occur with no more frequency than in premature children who do not develop the disease. The single apparent exception is hemangioma, the incidence of which is in the neighborhood of 5 to 20 per cent in many reported series. This is about the frequency in children whose eyes remain unaffected. Reese and Payne,[14] in 1946, suggested that the primary lesion of retrolental fibroplasia might be regarded as hemangioma, or as analgous to it, but there has been no support for this theory.

Krause's[9] idea that cerebral anomalies are part of the syndrome has also won no support. As a matter of fact, mental retardation is apparently no more frequent in premature children with the disease than in those without it. In 1950 Krause and his group[20] elaborated his original idea by suggesting that the disease might occur in two forms, one of prenatal origin, with 80 per cent or more of mental defectives, and a postnatal form, in which about half of the children are mentally retarded.

My own experience would not support such a concept. Moreover, if these children seem mentally retarded, there may be other explanations. I have had under observation since he was 3 months old a little boy who is now 4½ years old. When I first saw him, he had no apparent vision, he would not open his eyes in the light, and a typical gray reflex was present. As the disease progressed the anterior chamber became shallow, posterior synechias formed, the cornea was enlarged and cloudy, and secondary glaucoma developed and was so painful that the child cried almost incessantly. When he was 8 months old, on the indication of pain, I performed an iridencleisis on each eye, with almost immediate relief. His sight, of course, was irrevocably destroyed, and at the present time he has only light perception, in one eye. He has always been a difficult child and his adjustment at nursery school has been particularly poor. His teachers regarded him as mentally retarded, but since they have accepted my explanation that his apparent retardation is the result of psychologic trauma in the early months of life, and have taken that fact into account in their management of him, his adjustment has greatly improved.

The photophobia which these children present suggested light as a possible cause of their condition. Several observers have eliminated it by taping one eye, or both, immediately after birth. This practice did not prevent the disease but it did, incidentally, eliminate repeated ophthalmoscopic examinations as a possible cause.[6]

Some series, such as that of Speert and his associates,[5] show an abnormally high incidence of miscarriages and premature births in the past and subsequent histories of mothers of children with retrolental fibroplasia. This seems of no great significance, since there is an established family susceptibility to both abnormalities. Some series, particularly the one reported by Exline and Harrington[21] from the New Orleans Charity Hospital, show a rather high incidence of such maternal complications as pre-eclampsia, eclampsia, placenta previa, and premature separation of the placenta, but these are, in themselves, all causes of prematurity. Many series have not been studied from this standpoint; all should be. The ages of the parents, parental syphilis, Rh incompatibility, the prepartal management and medication

of the mother, and maternal nutrition have all been excluded.

No specific infective or metabolic maternal disease has ever been identified, though this seems the most reasonable of all hypotheses. The theory of a subclinical maternal infection has been advanced but not substantiated. Rubella has appeared in an occasional case, but it is well known that retrolental fibroplasia is not related to this disease.

Most observers consider parity a matter of no great significance, but Kinsey and Zacharias think that it may be important.[16] In their series the incidence of the disease was less than half (9.5 per cent) in first-born premature children as compared with children of succeeding pregnancies (19.7 per cent). One or two other observers have the same idea, but still others believe that the first child is more frequently affected.

As to the children themselves, it would seem that there is no possible cause which has not been suggested and eliminated, including methods of feeding; the time of weaning, if breast feeding has been practised; dehydration from withholding food for the first day or two after delivery; transfusions; possible vitamin A or vitamin E deficiency; the use of water-miscible vitamin supplements; and the use of iron. Silverman and his associates[17] are undoubtedly correct in their statement that no correlation can be established between the appearance, or the time of appearance, of ocular changes and the time of administration, duration of administration, or withdrawal, of medicinal iron, water-soluble vitamins, low-fat formulas, anti-bacterial drugs, or transfusion therapy. The disease has appeared in infants not treated by these measures and has not appeared, or has appeared and regressed, in infants treated by them. If the solution of the etiology of retrolental fibroplasia is along the lines of management of the newborn child, the key to the riddle has not yet been found, and that not for lack of intensive studies directed to this end.

The Role of Oxygen.—The only investigation along the lines of pediatric management which has led to any positive results has to do with the use of oxygen and the theory of anoxia.

Ingalls[22] seems to have been the first to advance the theory that sublethal oxygen deficits resulting from such conditions as antepartum hemorrhage, placenta previa or eclampsia might lead to permanent damage of the fetal vascular tissue. Experimentally, he obtained eye defects somewhat similar to the lesions of retrolental fibroplasia by subjecting rats to anoxic insults in the course of pregnancy. Szewczyk[23] was the first to suggest, and to advance proof for the suggestion, that retrolental fibroplasia is due to subclinical anoxia at a period of life when the incompletely developed retina is utilizing oxygen at a particularly rapid rate. His theory is that the vascular engorgement present in these cases is a compensatory mechanism, which does not appear when there is no lack of oxygen. If there is a lack, and it is not satisfied, the condition progresses to edema of the retina, transudation, hemorrhage, retinal separation, atrophy and fibrosis.

The proof advanced for this theory is persuasive. In 1951, when the duration of care of premature babies in oxygen had to be shortened at the Christian Welfare Hospital in St. Louis because of increased admissions, the incidence of retrolental fibroplasia rose alarmingly. Yet the records showed that the children who developed the disease had been kept in oxygen on an average of ten days longer than those who did not. Since the only criteria used to determine the length of the stay in the incubators were the color, breathing and activity of the child, it was concluded that the children kept in them longest were actually more oxygen-deficient than the others. There was no indication of ocular abnormalities until they were removed from the incubators.

Szewczyk's first report dealt with 9 children who presented vascular engorgement, tortuosity of the vessels, retinal hemorrhages and early retinal detach-

ment after they were removed from the incubators. Their eyes improved dramatically, sometimes within twenty-four hours, when they were replaced in oxygen, in a concentration of 60 to 70 per cent. The fundi of all of these children were eventually normal except for some residual tortuosity of the retinal vessels. The changes characteristic of retrolental fibroplasia were induced rapidly in 7 children who were removed from a 50 per cent concentration of oxygen in the incubators and placed in a 22 per cent concentration in bassinets. Within twenty-four to forty-eight hours dilatation of the retinal vessels was apparent. Within three to four days the vessels were tortuous, neovascularization occurred, and hemorrhages and progressive retinal separation also appeared. There was rapid reversal of the changes, usually within twenty-four hours, when the infants were placed in 60 per cent oxygen, and almost complete regression occurred within three to four days. In Szewczyk's [24] second report, which covered a larger number of cases, he also noted the effect of respiratory disease and the associated oxygen lack on the state of the eyes. In this report he carried his argument a step further by stating that retrolental fibroplasia can be caused by failure to withdraw oxygen gradually, to permit the child to become acclimatized to the lower oxygen concentration of the atmosphere.

The present opinion in regard to oxygen as a factor in retrolental fibroplasia is extremely confused. Bedrossian [25] largely supported Szewczyk's observations on the risk of rapid withdrawal of oxygen, the effect of respiratory disease, and the possibility of arresting the active vascular phase of the ocular changes by replacing the infant in oxygen. He does not regard oxygen as the direct cause of the disease but considers that the use of high concentrations over long periods of time may predispose to it. Two reports from Holland [26, 27] also confirm Szewczyk's observations, though in these cases regressive changes were less dramatic when the children were replaced in oxygen. Kinsey and Zacharias [16] observed that children with retrolental fibroplasia had been kept in oxygen for longer periods than those who did not develop it.

Numerous observers regard high concentrations of oxygen as predisposing to the disease. LaMotte, [28] in discussing Bedrossian's paper, stated that at the Delaware Hospital in Wilmington the incidence had been reduced from 1:3 (33.8 per cent) to 1:27 (3.7 per cent) in children weighing less than 4 pounds in the weight group predisposed to the development of the disease by using oxygen in the lowest possible concentration for the shortest period of time compatible with survival in infants. Ryan's [11] observations were to the same effect. Retrolental fibroplasia did not occur at the Woman's Hospital in Melbourne until after oxygen incubators had been installed and oxygen began to be given liberally to premature infants, whether they needed it or not. The disease disappeared after the use of oxygen was restricted to children who were actually cyanosed.

As the situation stands now, we must believe that both the use of oxygen and the lack of oxygen cause retrolental fibroplasia in premature children, and that its use also halts the progression of the disease and reverses the pathologic process which has occurred. What adds to the confusion in this theory, as in others which have been advanced, is that much of the evidence directly in conflict with other evidence, and vice versa, has been adduced by authorities eminent in the field of ophthalmology and of unimpeachable experience and probity.

Part of the difficulty arises from the fact that a great many published reports are lacking in important details. Part of it also arises from the fact that no single series is really large enough to be significant statistically. Finally, because of the varying conditions under which most of the studies were made, it is impossible to combine the various reports and analyze them as a total study.

The Cooperative Study of Environmental Oxygen and Retrolental Fibroplasia was set up July 1, 1953, in nineteen cooperating hospitals in various parts of the country, to overcome these difficulties, at least insofar as oxygen is concerned. The survey is being financed jointly by the Institute of Neurology and Blindness of the National Institutes of Health, the U. S. Public Health Service, the National Foundation for Eye Research, and the National Society for the Prevention of Blindness. It is under the chairmanship of V. Everett Kinsey, with the Kresge Eye Institute serving as the coordinating center, and is conducted by pediatricians and ophthalmologists in the cooperating hospitals. The purpose of the study is to determine whether the incidence of retrolental fibroplasia is dependent upon the amount of oxygen given the premature infant. A second purpose is to obtain information concerning the premature child and his parents which might be useful in the interpretation of the results of this study and in future investigations.

The investigation is limited to infants weighing 1,500 gm. or less at birth. The coordination center in Detroit is notified by wire of the birth of each premature child and the baby, by direction from the center, is given either a concentration of oxygen higher than 50 per cent for 28 days or as little oxygen as would be safe in the light of his condition. Concentrations of oxygen are checked regularly by specified methods. A tapering-off routine is followed when the child is removed from the incubator. With the exception of oxygen administration, the feeding plans, administration of vitamins (E and B are not given), chemotherapy, and other medications and management are the same in both groups.

The eyes are examined by a fixed technique and schedule, beginning as soon as possible after birth and continuing through the twenty-fifth week of life. As far as possible, ophthalmologists are kept in ignorance of the group to which the child under examination belongs. Retrolental fibroplasia is classified according to a standard scheme, and uniform records are provided for notations.

The following classification of retrolental fibroplasia is used in this study:

Active Phase

Stage 1. Dilatation and tortuosity of retinal vessels. Hemorrhages may or may not be present. Early neovascularization in the extreme periphery of the visible fundus may be present.

Stage 2. Stage 1 plus neovascularization and some peripheral retinal clouding. Hemorrhages are usually present. Vitreous clouding may or may not be present. Spontaneous regression may occur.

Stage 3. Stage 2 plus retinal detachment in the periphery of the fundus. Spontaneous regression is unlikely.

Stage 4. Hemispheric or circumferential retinal detachment There is elevation of the retina over a large area, but some of it is still in position.

Stage 5. Complete retinal detachment.

Cicatricial Phase

Stage 1. Small mass of opaque tissue in periphery of the fundus without visible retinal detachment. The fundus may have a pale appearance. The blood vessels may be attenuated.

Stage. 2. Larger mass of opaque tissue in periphery of the fundus with some localized retinal detachment. The disc is distorted by traction toward the side of the tissue mass, which is usually temporally located. Cases ending in stage 1 or 2 have useful vision.

Stage 3. Larger mass of opaque tissue in periphery incorporating a retinal fold which extends to the disc. Visual acuity varies from 5/200 to 20/50.

Stage 4. Retrolental tissue covering part of pupillary area. A small area of attached retina may still be visible, or only a red reflex over a sector of the fundus may be seen.

Stage 5. Retrolental tissue covering entire pupillary area. No fundus reflex is present.

One of the conditions of participation in this study is that the results in individual hospitals shall not be tabulated, published or discussed until the results of the whole study have been presented. I would therefore be in no position to discuss the data at the New Orleans Charity Hospital even if I were aware of the results, which I am not. What will come of this investigation it is not possible to say, except that the mass of material available and the controlled conditions of the study should make it possible to establish, one way or the other, the role of

oxygen in the causation of retrolental fibroplasia. Even if this particular problem is not solved, the method of the investigation should serve as an example for future studies along other lines.

DIAGNOSIS

The diagnosis of retrolental fibroplasia is not ordinarily difficult. Prematurity is the first clue. On the first examination the eyes are normal in size, the cornea and other structures, including the fundus, show no abnormality, and the anterior chamber is not abnormally shallow. The onset is shortly after birth, in most cases before the eighth week of life. The disease is characteristically bilateral, though one eye is often more severely affected than the other. The typical early findings are engorgement and dilatation of the retinal vessels, edema of the retina, small peripheral hemorrhages, and neovascularization, which is also a late finding. Other later findings include posterior synechias, detachment of the retina, a shallow anterior chamber, and secondary glaucoma. In the terminal stage, although the eyes may be grossly normal, they are often smaller than normal. The sockets are deepened, and the eyes seem sunken because of the loss of retro-orbital fat. If the child is not seen until he is 12 or 14 months old, he will show a distinctive tendency to rub his eyes with the backs of his hands.

Guy and his associates,[19] who state that abnormalities may be observed within the first few days of life, list as possible abnormal findings at this time a vitreous haze, myopia requiring a minus correction of 1 or more diopters on the ophthalmoscope, and dilatation and tortuosity of the retinal vessels. Many would not agree with all of these criteria and would regard haziness of the vitreous, enlargement of the retinal veins, and a grayish coloration of the retinal periphery as normal findings in very young children.

The differential diagnosis, while it may include any disease characterized by disorganization of the retina and vitreous, chiefly concerns persistent hyperplastic vitreous, retinal dysplasia, and retinoblastoma.

Persistence of the hyperplastic vitreous is uncommon. It is usually bilateral. The involved eye is often somewhat smaller than the normal eye. The opaque tissue behind the lens is densest in the central portion and fades away in the periphery, where long ciliary processes are invariably seen. If the opaque tissue behind the lens is sufficiently thin about the periphery to permit a view of the interior of the eye, a persistent hyaloid artery can usually be demonstrated.

Retinal dysplasia is also uncommon. It is always bilateral. It occurs in the newborn but it is not related to prematurity. The eyes are usually microphthalmic and there are associated malformations, chiefly of the heart, vascular system, or skeleton, or of the brain, with mental retardation. One or the other of these conditions frequently causes early death. Histologically the retina shows signs of dysplasia and often of rosette-formation. The fibrous tissue behind the lens seems to be persistent primary vitreous. Secondary vitreous is usually scanty if it is formed at all.

Retinoblastoma usually occurs in full-term infants and does not become evident until the end of the second year. It is primarily unilateral. The parents usually bring the child for consultation because they have noted a gray reflex in the fundus. The affected eye tends to cross, but there are no other external abnormalities. The anterior chamber, iris and lens are all clear. In the fundus is an irregular mass, which sometimes can be seen grossly. Retinal detachment is frequent. The differential diagnosis of retinoblastoma is essential, for it is an extremely malignant tumor and prompt enucleation is indicated, whereas this operation is never indicated in retrolental fibroplasia, at least for the primary disease.

Ophthalmoscopy.—Retrolental fibroplasia will not be recognized until it has reached an advanced stage if no search is made for it until it becomes clinically evi-

dent. The early stages are demonstrable only by the ophthalmoscope, and examinations must begin as soon as is practical after birth and must be carried out serially, at regular intervals. The best plan is to examine the child weekly until it is three months old. Then, if there is no evidence of the disease, the examinations are made monthly. If pathologic changes are apparent, they are made at least once a week. Some observers believe that ophthalmoscopy may be safely discontinued at three months, in the belief that if the condition is to develop there will be evidence of it by this time. This is not a safe plan. In a number of reported cases, as well as in my own experience, the first manifestations of the disease appeared after the third month. The examinations should therefore be continued until the child is at least six months old, and my personal preference is to continue them for nine months.

This is, of course, more easily said than done. The ophthalmoscopic examination of a young child is often a real ordeal, aside from the fact that the average premature nursery does not have the facilities which would simplify the procedure. At the best, a thorough examination with the electric ophthalmoscope consumes twenty to thirty minutes. Unless the periphery and posterior segment are included, early changes as well as residua may be overlooked.

Good dilatation of the pupils is essential for a successful examination. We prefer to use 2 per cent homatropine hydrobromide and 10 per cent neosynephrine (phenylephrine hydrochloride), one drop in each eye. Dilatation is adequate within the hour, even when the iris is heavily pigmented. Examination is almost impossible in the incubator and the best plan is to remove the child and wrap him securely in a blanket while it is carried out.

General anesthesia is occasionally necessary, particularly as the child grows older, but it can usually be avoided by a few common sense precautions. Our own plan is to instruct the mother not to feed the child until he is brought to the office. He is then fed, and is usually quiet for a long enough time thereafter to permit the examination to be carried out.

PROPHYLAXIS AND THERAPY

Not much time need be spent on either the prophylaxis or the therapy of retrolental fibroplasia. There is no prophylaxis except for the obstetrician to use every means at his command to forestall premature delivery. He would do this in any event, but the responsibility is heavier at the present time in the light of what we know may happen to premature children, and will inevitably happen to a certain proportion of them. In the present state of the oxygen controversy it also seems the part of wisdom to use oxygen with discretion, avoiding excessive concentrations, giving it only when it is necessary, giving only as much as is necessary, and weaning the child from it by withdrawing it gradually.

There is not much more to be said for therapy. X-ray therapy is useful in the control of vascularization but it has given no results in retrolental fibroplasia. Corticotropin and cortisone seemed logical, on the ground that hyperadrenalism inhibits the growth of the capillaries and that the premature infant, because of prematurity, is deficient in the hormones of the adrenal cortex during its early postnatal life. The original results seemed promising, but further observation altered the picture. Reese,[29] in fact, stated that he had obtained specimens from two patients in whose deaths ACTH had played an important, if not the chief, role. One of the worst cases of retrolental fibroplasia which I have ever seen was treated by this agent by the intramuscular and subconjunctival routes, and I have long since discontinued its use.

Excision of the retrolental mass was practised in some of the earlier cases, but the operation was abandoned when it was found that the excised mass was composed largely of retinal elements. Reese and Blodi[30] reported operating on 52 eyes,

in an attempt to remove enough tissue in the pupillary area to permit the use of any partially attached area of the retina. In 27 cases the condition was the same after operation and in 16 cases it was worse. In the remaining cases, although there was some objective improvement, there was no certainty of functional improvement in any instance. All of the excised tissue contained retinal elements, which were often disorganized and degenerating. Neither this technique nor any other surgical procedure which has been attempted seems rational, and none of them has proved useful. Surgery for glaucoma, however, may be necessary if the eyes become intractably painful. It should always be a last resort.

At the present time the only therapy at all worthwhile seems to be the use of mydriatics, to prevent or control synechia formation, which, in turn, may prevent secondary glaucoma. My own preference is for drops of atropine and scopolamine. The inflammatory reaction is often severe enough to occlude the pupillary area completely, and this medication, if the child has any degree of retinal function, may give him a pupil which is open and which he can see through.

In the evaluation of any form of prophylaxis or therapy which may seem to give results, it must be remembered that retrolental fibroplasia is a self-limited disease, which may regress naturally, and that it is characterized by unpredictable fluctuations of incidence. It is entirely possible that the results which may seem to have been accomplished by therapy may be explained by these reasons.

JOINT RESPONSIBILITY FOR RETROLENTAL FIBROPLASIA

At the present time retrolental fibroplasia is a matter of great concern in teaching hospitals and research centers. Otherwise, there is no general sense of responsibility about it. One reason for this attitude is the feeling of many obstetricians and pediatricians that since therapy is so ineffective, the mere identification of the disease serves no useful purpose. Another reason is the expense connected with such investigations. The question of fees cannot be ignored in private practice.

A final reason for failure to carry out routine ophthalmoscopy on all premature children in private practice is the cumbersomeness of pediatric-ophthalmic consultation. Benton's [31] plan simplifies this aspect of the problem. He proposes that in private hospitals each pediatrician designate, in advance, the ophthalmologist whom he wishes to examine his premature babies, so that each child may come under ophthalmologic care as a matter of routine as soon as the necessary consent has been obtained from the parents. This is a simple and sensible plan, which all obstetricians and pediatricians would do well to adopt, since they are the ones who must take the steps which bring the ophthalmologist into the case. At the present time there is not much that he can do beyond diagnosing or eliminating the disease, but it is inconceivable that some technique will not eventually be detected to control this grave complication of prematurity.

SUMMARY

Retrolental fibroplasia, which was an unknown disease up to 1942, is now the commonest cause of blindness in young children. Its incidence has increased enormously in the last several years. The etiology is unknown beyond the fact that it is a disease of prematurity. There is no known method of prophylaxis or therapy. The most hopeful aspect of the disease at the present time is that pediatricians and obstetricians have come to realize that it is their responsibility also. The ophthalmologist does not enter the case until they call him in. It would be a wise plan to set up a system in all hospitals in which such consultations would be routine.

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OFFICERS OF THE LOUISIANA STATE MEDICAL SOCIETY FOR 1954 - 1955

At the Seventy-fourth Annual Meeting of the Louisiana State Medical Society, new officers were elected. Considering the widespread activities and responsibilities that are part of organized medicine today, it is fortunate to again have a group of physicians in office who are capable and willing to uphold the standards and carry on the traditions consistent with the illustrious history of our Society.

Dr. Walter Moss, widely known surgeon of Lake Charles, is President for 1954-1955. His well rounded professional training included interneship at Charity Hospital of Louisiana at New Orleans, followed by a House Surgeonship for four years. He is a fellow of the American College of Surgeons and a diplomate of the American Board of Surgery, as well as a fellow of the International College of Surgeons. His work in the field of organized medicine has extended over many years. He is a former First Vice President of the State Society, and was President of the Calcasieu Medical Society during 1936-1937. He has served the State of Louisiana as Chairman of the State Board of Institutions in 1941-1942, and his City, Lake Charles, where he is now on the Board of Directors of the Association of Commerce. He was President of the Tulane Alumni Association in 1946-1947. We feel sure that he will not only carry on our best traditions, but in these changing times, when tradition alone does not suffice, will wage the good fight and advance the cause of organized medicine.

Dr. Max M. Green, prominent urologist of New Orleans, becomes President-elect. Dr. Green interned at Charity Hospital, New Orleans, and then served as Resident in Urology during 1930-1932. He is a fellow of the American College of Surgeons and a diplomate of the American Board of Urology. He has been for many years active in the affairs of the Orleans Parish Medical Society, and was a member of its Board of Directors from 1940 through 1949, during which time he held the positions of Secretary, 1st Vice President, 3rd Vice President, Librarian, and in 1948 was President. He has also been Secretary of the New Orleans Graduate Medical Assembly. He is active not only in the field of organized medicine but in that of medical education also, as Assistant Professor of Urology at L.S.U. Medical School. His civic services include the Chairmanship of the Medical Committee of the Chamber of Commerce of the New Orleans Area. The Society is fortunate in having chosen a physician of his calibre as President-elect.

Dr. Henry W. Jolly, Jr., Baton Rouge, 1st Vice President, has been active in the East Baton Rouge Medical Society and was President of that Society. During the past year he has been Chairman of the Committee on Public Policy and Legislation. He has served with success and distinction in the handling of the affairs of this important committee, and is due special credit for his success in defeating the efforts of the chiropractors to secure a State license this year.

Dr. George H. Hauser, 2nd Vice President, is a distinguished pathologist of New Orleans, who has served the State Society in many capacities over a period of years. He has been active in the affairs of the Parish and State Societies, particularly on committees pertaining to pathology and public health, and has been alternate to the House of Delegates of the A. M. A. on many occasions.

The 3rd Vice President is Dr. W. A. K. Seale of Sulphur, who has worked hard and diligently in the affairs of Calcasieu Parish for many years. His energy and judgment have been useful in organizing opposition in the 7th Congressional District to the campaign of the chiropractors. He will add further stability to our councils.

Dr. C. Grenes Cole, Secretary-Treasurer, continues his term of office, and our appreciation of his experience and counsel grows with the years. His resourcefulness and energy evoke the admiration of the active members of the Society.

Chairman of the House of Delegates is Dr. W. Robyn Hardy, who enters his second year in this capacity. Although presiding for the first time this year, his expeditious and fair handling of parliamentary problems was a source of gratification.

He was materially assisted by Dr. Ralph H. Riggs of Shreveport, Vice-Chairman, whose good judgment and experience have meant much to the cause of organized medicine.

Dr. J. E. Clayton of Norco succeeds Dr. Joseph S. Kopfler, who retired as Councilor from the Second District after many years of service. Dr. Clayton has been very helpful in the organizational activities of the Second District Medical Society, and equally valuable to the Legislative Committee. The remainder of the Council is unchanged, and its personnel continues to be a source of satisfaction to the membership.

As the problems of organized medicine increase, the responsibility and urgency of the Society's affairs become a more serious burden. The profession of the State may rest assured that the administrative organization which they have chosen at the Seventy-fourth Annual Meeting is composed of capable physicians upon whose judgment and experience they can rely and whose leadership they can trust, not only in State matters, but in the fight against the ever encroaching efforts of federal control of medicine.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

LOUISIANA LEGISLATURE

The Legislature is in session, and the expected chiropractic bill has been introduced under House Bill 111 (by Fields, et al) to regulate the practice of chiropractic and create a board of chiropractic examiners.

This bill will, in all probability, be referred to the Committee on Public Health and Quarantine, composed of the following members: Charles E. Deichmann, Chairman, Orleans Parish; James J. Villemarette, Vice-Chairman, Avoyelles Parish; Cecil R. Blair, Rapides Parish; Mrs. Bland

Cox Bruns, Orleans Parish; Geo. E. DeVille, Orleans Parish; Richard D. Guidry, Lafourche Parish; Reeves Morgan, East Feliciana Parish; Mrs. Richard S. Parrott, St. Landry Parish; Lonnie Richmond, West Carroll Parish and Ford E. Stimon, Bossier Parish. We think Woodrow Wilson, Morehouse Parish, who defeated A. P. Carter, will be the eleventh member of this committee.

You members who reside in these respective parishes should contact these representatives, best in person, or by telephone, telegram or letter immediately, asking that they support us in our efforts to protect our citizens by defeating House Bill 111, which would legalize these cultists to practice medicine in Louisiana.

We are depending upon you to do your duty, so do not let us down.

CONGRESSIONAL BILLS

We feel that the membership would like to know what is being done in Washington regarding two of the most important bills affecting organized medicine before Congress today. These bills are H.R. 8356 and H.R. 7199.

Following are two statements which are being presented before Congressional Committees by representatives of the American Medical Association. These bills deal with the President's "Re-Insurance" proposal, extended coverage under the Social Security Act to include physicians.

"I am Dr. David B. Allman of Atlantic City, New Jersey, where I am engaged in the practice of medicine. I am a member of the Board of Trustees and the Chairman of the Committee on Legislation of the American Medical Association. I am appearing here today as a representative of that Association concerning H.R. 8356, 83rd Congress.

"We are grateful to the Committee for permitting us to delay our appearance on this bill until after the joint meeting of our Committee on Legislation and the Executive Committee of the Board of Trustees which was held in Chicago last Wednesday, March 31. Our organization has now formulated a position on the bill which I shall present today.

"Inasmuch as you have been conducting hearings on this measure for some time, it is unnecessary in our statement to review the bill in detail. I should like to state at the outset, however, that the American Medical Association is in complete accord with the principal purpose of the bill, which is to promote the best possible medical care on reasonable terms. It is reassuring to the medical profession to find that the official position of the government is one of trust and confidence in the ability of private initiative to solve existing problems in the field of medical care. As was stated before this Committee last January, our Association has for many years adhered to a policy which parallels the above referred to purpose of this bill. We are also in agreement that the most feasible method of accomplishing this result for most of the people is through voluntary health insurance.

"The Association, however, seriously doubts whether the mechanism suggested in H. R. 8356 is essential and whether it will, in fact, accomplish the desired results.

"In determining its essentiality we believe that it is necessary to give full and complete consideration to the tremendous strides which voluntary health insurance has made in this country and the simultaneous improvement in benefits provided to meet the desire of the public for more adequate protection. The expansion of coverage and the improvement of benefits to cushion the economic shock of hospital, surgical and medical expenses, has been phenomenal during the past few years. Inasmuch as complete statistical data outlining this expansion and growth have been made available to your Committee by previous witnesses, I shall not repeat these figures at this time. I understand you have also been briefed on the progress and prospects of the newest area of health insurance — 'major medical expense' coverage which is designed to provide protection against the unpredictable,

the unexpected, illness, acute or chronic, the financial impact of which would seriously disrupt the family budget.

"It is our belief that the figures which have been presented hold real promise for still greater progress in health insurance coverage. This fact plus the demonstrated ability of the industry to meet the needs and demands of the public indicates to us that it is unnecessary for the federal government to enter the field. The public interest will be served best and continued progress of health insurance protected by preserving the freedom and competitive features of the present method of operation.

"It must also be emphasized that there is a limit to the number of insurable people in this country, and a limit to what can be accomplished by insurance. In any effort to solve the economic problems of medical care, it is essential to consider two groups of individuals: (1) those who are able to pay the normal costs of medical care, and (2) those who are indigent. Most of the American people fall within the first group and have access to the many forms of health insurance now offered. Some of these have elected to carry their own risk either because of their financial ability to do so or because they are not convinced of the wisdom or necessity of purchasing protection against medical expenses in advance. As the desirability of insuring against medical expenses is more generally accepted and as the improvement and development of new types of coverage evolve, it is reasonable to expect that the maximum number of 'insurables' will be covered.

"The other group—the indigent—poses distinctly different problems. These individuals do not have the funds with which to purchase insurance and are dependent, in some measure, on outside assistance for the basic necessities of life. The American Medical Association believes that if the medical care problems of this group cannot be solved by the individuals or their families, the responsibility should be assumed by the local and state government.

"As an Association we have been greatly concerned with the individuals in this category and are making efforts to aid in the solution of this very difficult problem. Through our Council on Medical Service we have undertaken a series of studies of the organization and operation of state and local indigent medical care plans which are effective in meeting the need. Fifteen such studies have been made to date and five more will be completed this summer. These reports are being published in *The Journal* of the Association and distributed to all state and county medical societies. Through this method and through the establishment of suggested criteria for such plans medical societies have been stimulated to organize an adequate program or improve an existing one.

"Recognizing that the medical profession is only one of a number of parties concerned in the total health care picture of the indigent a permanent study group comprised of representatives of the American Medical Association, the American Hospital Association, the American Dental Association, the American Public Health Association and the American Public Welfare Association has been established. Two immediate projects being undertaken by this study group are:

(1) Preparation of a basic statement on the total problem of health care for the indigent.

(2) A series of joint field studies by staff personnel of the various agencies represented so as to consider the problems, operations, and services of all groups concerned with providing medical, hospital and related services to the indigent.

"Finally, the Association is considering the establishment of a field service to assist state and county medical societies in developing programs in this field. As a pilot project along this line, the field staff spent five weeks in one state during the summer of 1953 to help collect basic data and information which might lead to a

formal state organization and to adequate financing for an indigent medical care program.

"We hope that expanded efforts by private agencies and an awakening by state and local governments to their responsibilities to the indigent will lead to a solution to the problem. It is not clear, however, how persons in the indigent group will be assisted by the provisions of the bill under consideration, without some form of federal subsidization paid through the insurance companies. We believe this would be objectionable.

"As physicians, we have a real interest in this bill; nevertheless, we do not feel qualified to comment authoritatively on its technical insurance and reinsurance aspects. We have, therefore, in considering the bill, met with representatives of the insurance industry and have studied carefully their testimony before this Committee during the past week. We have noted the reluctance and in some instances, the expressed fear with which these insurance authorities have viewed the proposal.

"We were impressed by the comments of Mr. Edwin J. Faulkner when he said, in testifying before your Committee on March 26:

> 'Government reinsurance of health insurance plans would introduce no magic into the field of financing health care costs. Reinsurance can distribute risks among insurers just as insurance distributes them among policyholders, but no matter how far this distribution is carried, it must be sound to succeed. Reinsurance does not increase the ability of the insurer to sell protection to the unwilling buyer. Reinsurance does not reduce the cost of insurance. Reinsurance does not make insurance available to any class of risk or geographic area not now within the capabilities of voluntary insurers to reach.'

"Another statement by Mr. John H. Miller on behalf of three health and accident insurance associations with over 300 member companies stated, in part:

> 'Reinsurance, therefore, does not provide a means of making insurable what would otherwise be an uninsurable risk. It does not add to the aggregate resources of the insurers. It does not help to sell insurance nor does it reduce the cost of insurance. If our citizens are not to labor under a misunderstanding, it is essential for them to realize that reinsurance is not a panacea, and that it does not provide additional funds to finance the cost of medical care. As has been made clear in this presentation of the program, voluntary insurance cannot relieve the community of the burden of providing medical care to the indigent nor is it helpful to those who presently cannot qualify for it, and reinsurance does not enhance the power of insurance in these areas. The reinsurance facilities have been proposed with the purpose of extending the frontiers of insurance effectiveness. The rapid development of accident and sickness insurance has, however, been accomplished with little use of the already extensive reinsurance facilities available to the insurers, and we see no evidence that progress would have been augmented had reinsurance been used to a greater extent. The underwriting, or risk bearing, capacity of the companies in this business is very large.'

"These and other expressions of opinions from the insurance industry plus our own analysis of the bill have led us to the conclusion that a federal reinsurance system such as proposed by the bill could not be expected to achieve the objectives set forth in this legislation. In addition, the measure as drawn would place extensive regulatory power in the Secretary of the Department of Health, Education and Welfare. The concentration and delegation of such potential power and control over a vital branch of American industry in a department of the Executive Branch of the government without clear and convincing evidence of need is extremely difficult to justify.

"Let me reiterate that we believe the sponsors of this proposal are deserving of

commendation in attempting to spread voluntary health insurance through private initiative. Nevertheless it is the belief of the American Medical Association that the bill will not fulfill its intended purpose and may, in fact, inhibit the satisfactory progress which is now being made by voluntary insurance companies. For these reasons it is the recommendation of the Association that this bill should not be reported favorably by this Committee.

"In conclusion, let me express my appreciation for myself and for the American Medical Association in permitting an expression of our views on this bill."

"I am Dr. F. J. L. Blasingame, of Wharton, Texas, where I am engaged in the active practice of medicine. I am a member of the Board of Trustees of the American Medical Association and am appearing today on behalf of that Association in connection with H. R. 7199, 83rd Congress, currently being studied by your Committee.

"My testimony is directed to only two aspects of the bill, namely, the suggested compulsory coverage of physicians under Title II of the Social Security Act, as amended, and the 'waiver of premiums' provision included in section 106. The Association has not taken any position with regard to the other provisions of the bill which do not have primary medical implications.

"The American Medical Association strongly opposes compulsory coverage of physicians under Title II of the Social Security Act. In June 1949 the House of Delegates adopted a resolution to this effect. Another such resolution was adopted at the last meeting of the House of Delegates, in December, 1953. The most recent resolution adopted in December, 1953 provides:

'Whereas, Legislation has been introduced into the Congress providing for the extension of compulsory coverage under Title II of the Social Security Act to self-employed persons, including physicians; and

'Whereas, The House of Delegates has in the past registered its disapproval of the principle involved in this legislation; and

'Whereas, Physicians desire the continued right to establish retirement benefits for themselves on a voluntary basis rather than being compelled to participate in a tax-supported government program which they do not want; and

'Whereas, The Jenkins-Keogh bills (H.R. 10 and H.R. 11) now before the Congress provide for development of a voluntary pension program which is equitable, free from compulsion, and satisfies the retirement needs of physicians; therefore be it

'Resolved, That this House of Delegates reaffirm its support and endorsement of the voluntary pension program provided in the Jenkins-Keogh bills and its strong opposition to the extension of compulsory coverage of physicians under Title II of the Social Security Act'

"In February, 1954 the Board of Trustees again reviewed this question and reaffirmed the Association's position of opposition to compulsory coverage. The Board did determine that no objection would be raised to the extension of the Old Age and Survivors Insurance provisions of Title II so as to permit voluntary coverage of physicians.

"We consider it absolutely incompatible with the free enterprise system for a group to be compulsively covered under a governmental system of old age benefits when that group strongly and with great force opposes such coverage.

"We have carefully reviewed the report of the advisors to the Secretary of the Department of Health, Education, and Welfare and have been unable to find any reasons to justify this part of their recommendations. I am here to assure you gentlemen that the members of the medical profession do not feel discriminated against by having been excluded from the

provision of Title II of the Social Security Act. On the contrary, we believe that we are capable of planning for our security in old age and are not desirous of governmental intervention.

"Another and very practical consideration is the fact that only a small proportion of the self-employed physicians consider age 65 as a line of demarcation between their working years and retirement. Those who are able to work prefer to keep right on taking care of sick people; many of them taper off the amount of work they do as they grow older. However, in an unpublished study prepared by our Bureau of Medical Economic Research, it was found that one half of the physicians actually retiring from practice after age 65 did so after age 74.

"If forced under this program, the typical physician would be required to continue to pay OASI taxes from age 65 to age 74 or 75 before he would receive any benefits.

"This prospect of indefinite retirement for the members of another great profession was very well stated in a recent issue of the American Bar Association Journal (February, 1954) by Judge Allen L. Oliver, Chairman of the Committee on Unemployment and Social Security of the American Bar Association. I quote:

> 'In summary, the self-employed professional differs from the employed person because he is not forced into abrupt and complete retirement, because he usually continues substantially remunerative activities after age 65, because his entire life and training emphasizes individual activity rather than group treatment.'

"Old age benefits under the Act simply do not fit the economic pattern of the life of the self-employed physician. To compel him to come under the provisions of this portion of the Social Security Act would represent a failure to understand that physicians, like many other professional people, serve the citizens of their community best by following their traditional economic pattern of life. In making this comment, it is not my intention to make any inferences whatsoever regarding the applicability of Old Age and Survivors Insurance Program to the 50 million employed persons in the United States.

"I do not come here empty handed. The American Medical Association has repeatedly expressed its support of the Jenkins-Keogh bills which are now before this Committee for consideration. We are grateful to the Chairman, Mr. Reed, for having sponsored with Mr. Keogh the original bills of the previous Congress and to Mr. Jenkins, Mr. Camp, as well as Mr. Elliott and Mr. Keogh. These bills would provide a long-range plan for encouragement through tax-deferments for self-employed people to set aside limited amounts from their earned income into restricted retirement annuity or pension trust programs from which they could draw benefits on attaining age 65.

"I believe all members of this committee understand that these bills would provide only tax deferment, not tax avoidance. It would give the 11,000,000 self-employed people who want to save for their old age an opportunity to do so under conditions approximately equal to those provided by corporate pension plans approved by the Bureau of Internal Revenue under Section 165 (a) of the Federal Internal Revenue Code. H. R. 10 and 11, 83rd Congress, provide flexible arrangements which would fit the economic pattern of the life of the self-employed physician. It would encourage approximately 11,000,000 self-employed people to provide for their own retirement. I am glad to note that these identical bi-partisan bills would also grant tax-deferment encouragement to approximately 30,000,000 pensionless employed people. We support these bills heartily because they would provide prepaid pensions for all who are willing to save. We are glad to join hands in support of these measures with many other associations such as the American Bar Association, the American Dental Association, the American Institute of Accountants, and the American Farm Bureau Federation, many

of whose members are self-employed farmers. We believe that our country is so diversified and that people earn their living under so many different conditions that it is wise public policy for the Congress to provide a flexible pension system for the self-employed and to permit the pensionless employed to participate in the same program.

"The second portion of the bill concerning which I would like to comment is section 106 which proposes a waiver of premiums and the preservation of insurance rights of individuals with extended total disability. While we are pleased to note that this section of the bill includes a number of safeguards which did not appear in the bills which heretofore have been before this Committee, we are still constrained to oppose this portion of the bill because it most definitely would become an entering wedge for the regimentation of the medical profession by creating the mechanism for the adoption of a federal cash permanent and total disability benefit program, which in turn could lead to a full fledged system of compulsory sickness insurance. The provisions in this bill cannot be appraised solely as an isolated, detached effort to provide some measure of aid to the disabled worker. We believe that this and every other step in the direction of a compulsory sickness insurance system must be opposed.

"It is true that under the present system a man who is permanently or totally disabled or who goes from covered to non-covered employment during his lifetime is penalized. This is due to the fact that all of the years following the entrance of a wage earner into the Social Security System are counted as "elapsed years" and are used in determining his annual average wage, which is the key in figuring the basic Old Age and Survivors Insurance benefit. It is our recommendation that in lieu of the approach embodied in Sec. 106 the Committee give consideration to the more liberal formulae of most modern systems for computing benefits, such as those followed by most corporate pension plans and indeed by the government itself in the case of the Civil Service Retirement System. These systems involve the use of the five or ten best years during a man's working lifetime in computing retirement benefits with an allowance in the form of increment years for each year of gainful employment. This latter provision compensates a wage earner who has had a long period of covered employment and consequently has made larger contributions into the pension fund. If such a method were adopted, it would be unnecessary to consider gaps in a man's wage record, regardless of whether they were due to permanent or total disability, non-covered employment or any other cause.

"For the reasons which I have outlined above, we are opposed to the two provisions of H. R. 7199, 83rd Congress, to which my remarks have been directed."

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MEDICAL NEWS SECTION

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
Calcasieu	Fourth Tuesday every other month	Lake Charles
East Baton Rouge	Second Tuesday of every month	Baton Rouge
Morehouse	Third Thursday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

SEVENTH ANNUAL MEETING AMERICAN ASSOCIATION OF BLOOD BANKS

The Seventh Annual Meeting of the American Association of Blood Banks will be held September 13-14-15, 1954, at the Shoreham Hotel, Washington, D. C. In addition to an excellent scientific program, there will be interesting exhibits, a special course for technologists, and roundtable and panel discussions led by national and international authorities. For further information, write Office of the Secretary, American Association of Blood Banks, 3500 Gaston Avenue, Dallas, Texas.

CIVIL DEFENSE TRAINING COURSE FOR DENTISTS AT FCDA STAFF COLLEGE, OLNEY, MARYLAND, July 21-23, 1954

A special three-day course for dentists will be given July 21-23, 1954, inclusive, at the FCDA Staff College in Olney, Maryland.

This course for dentists is part of the FCDA training program for services vital to the civil defense sphere of national defense. Dentists are expected to give valuable auxiliary medical service in a major disaster inasmuch as the number of available physicians and nurses would be far too few to provide even emergency medical relief. This new course will afford dentists an opportunity to become familiar with the scope and nature of the civil defense home-front preparedness program and the role they will be called on to play.

The course will consist of lectures and demonstrations on orientation, organization and training, analysis of problems, tactical operations, and special dental relationships to civil defense. The effects of atomic and chemical warfare will be graphically presented. The class will also observe the actual training of rescue and firefighting services.

There is no charge for tuition. A charge of five dollars per day will be made to cover expense of board and room. Comfortable living quarters and meals are provided for the class members only, on the college grounds which are about 20 miles from Washington, D. C.

Application should be made promptly, as the number to be admitted to this first class is limited. The deadline for such applications is July 1. Applications should be sent in promptly to the Civil Defense Staff College, Olney, Maryland.

EMOTIONAL FACTORS MAY CONTRIBUTE TO EAR DISORDER

Emotional upsets may contribute to a form of deafness known as otosclerosis, according to Dr. Edmund P. Fowler, New York.

Writing in the January 23, 1954, Journal of the American Medical Association, Dr. Fowler stated that he has found the incidence of the disease—a formation of bone in the inner ear that results in deafness—to be greater in emotionally sensitive or unstable persons predisposed to the disease than in more emotionally stable ones.

"In otosclerosis the onset of deafness and its periods of rapid increase appear to coincide in time with, or more commonly subsequent to, puberty, childbearing, the onset of the menopause, severe illness, psychic traumas, and stress and anxiety, all of which are accompanied by major autonomic nervous system readjustments or dislocations," Dr. Fowler said.

HOUSEWIVES MOST PRONE TO SKIN DISEASES OF THE HANDS

Being "just a housewife" can be a dangerous occupation—one of the most dangerous, in fact, as far as skin disturbances of the hands are concerned.

Dermatitis of the hands gives rise to an immense amount of suffering and constitutes about nine per cent of the daily medical endeavor of a practicing dermatologist, it was stated by Dr. Richard L. Sutton, Jr., Kansas City, Mo., and Dr. Samuel Ayres, Jr., Los Angeles.

"The usual patient is a young matron, who must keep house, cook, wash dishes, do the laundry,

raise her children, and hold her husband; who can ill afford to sustain the misery of manual disability and whose occupation is inherently one of the most hazardous with which the dermatologist commonly has to deal," the doctors wrote in the current Archives of Dermatology and Syphilology.

Causes of inflammatory skin conditions of the hands are numerous, and to effect a cure it may be necessary to eliminate more than one condition, according to the doctors, who added:

"The skin as a whole is the dividing line between the person and his environment. It combines the features of a barrier, an adaptive mechanism, and a sensory organ—a great deal is asked of it, so to speak.

"The skin of the hands is exposed to the potentiality of diseases provoked by heat, cold, light, moisture, irritant chemicals, sensitizing chemicals, and pathogenic organisms to a degree over and above other skin. It is subject additionally to all the diseases of internal origin that skin is elsewhere, and the hands are especially subject, being special tactile organs, to psychosomatic disturbances."

There are many mechanisms provoking inflammatory skin disease of the hands, the doctors pointed out.

REHABILITATION CENTERS

The first authoritative manual of information on the nation's rehabilitation centers has been published by the National Society for Crippled Children and Adults, the Easter Seal Society, Lawrence J. Linck, executive director, announced.

"Rehabilitation Centers in the United States," one of the most useful recent publications in the field of service for the crippled, grew out of the First National Conference on Rehabilitation Centers jointly sponsored by the National Society and the Office of Vocational Rehabilitation of the U. S. Department of Health, Education, and Welfare.

The booklet was compiled by Henry Redkey, consultant on rehabilitation centers for the federal department.

TAKE BLOOD FROM SITTING DONOR, BLOOD BANK ADVISED

Two St. Louis blood experts have advanced the idea that it might be better for blood donors to give blood sitting up, rather than lying down as is usually done.

The suggestion was made in a paper delivered by R. O. Muether, M. D., and B. Koster, B. S., before the American Association of Blood Banks, at its sixth annual meeting in Chicago.

In a test involving 190 donors, 85 sat up and 105 lay down, Dr. Muether reported. Careful, repeated checks of blood pressure, pulse rate and incidence of reaction showed no appreciable difference, it was found. When questioned afterward, 54% of those who sat up and who had had previous bleedings lying down, said they preferred to sit up. Only 8% preferred lying down.

ADULT UNDERSTANDING OF CHILD ACCIDENT PROBLEMS NECESSARY

It's the adult's job to prevent childhood accidents.

As children do not always conform to the practices that would insure their greatest safety, adults must be made to understand the accident problem of children and must be as concerned about it as they are about childhood diseases, it was stated editorially in the January Journal of the American Medical Association.

"A notable decrease in infant mortality and in the death rate from childhood diseases has been brought about by the nation's physicians during the last half century," the editorial pointed out. "However, despite the efforts put forth by the medical profession to protect the country's future citizens, childhood mortality continues high because of accidents—accidents that, for the most part, are preventable.

"The accident death rate among children is being reduced only about one-third as fast as the rate for death by disease. Preventable accidents are the leading causes of death in the age group of 1 to 14 years, killing more children than the combined mortality of the next six causes of death—pneumonia, congenital defects, cancer, tuberculosis, leukemia, and heart disease—and killing many, many more children than poliomyelitis."

CARE AND PLANNING CAN SALVAGE RETIREMENT PRODUCTIVITY WASTE

With greater care and planning, much valuable productivity that now is being wasted by compulsory retirement can be salvaged, it was stated editorially in the current (January 16) Journal of the American Medical Association. Older workers who are capable of and desire employment should be permitted to work, it added.

As firms take great pains to choose whom they will hire, there is no reason why they should not concern themselves equally with the problems of whom they will retire, the editorial said. Retirement policies, in addition to setting the conditions of retirement, should state how older workers may be profitably kept on the job and how their status is to be determined.

The best way to determine who should be retired, according to the editorial, is for a firm to create a panel to judge each case on its merits and determine whether the worker should continue in his present status, go on a modified schedule, transfer to a less demanding job, or be

retired. The panel, which should include one or more high level executives and an industrial physician, should seek advice, when necessary, from the employee's immediate supervisor, the company's personnel director, or the local union. Restudying of methods of work and a restudying of training methods also will aid in the solution of the problem, it was added.

WOMAN'S AUXILIARY TO THE LOUISIANA STATE MEDICAL SOCIETY

JEFFERSON DAVIS PARISH

Women's Auxiliary to the Jefferson Davis Parish Medical Society have just completed one of the most active years ever experienced by them under the able leadership of their president, Mrs. L. E. Shirley, Jr. At their first meeting in October, a member gave an interesting and informative talk on Oveta Culp Hobby, the head of our recently created national Department of Health, Welfare and Education, dwelling especially on the amazing number of things "Mrs. Secretary" has accomplished, her rare talent for administration, and the heavy responsibilities she now carries.

In November, the auxiliary was honored by a visit from the State president, Mrs. Edwin A. Socola, who told the members present of the aims and objects of the state and national auxiliaries. Mrs. Socola was entertained at a luncheon.

In December, the members had a guest speaker who spoke on an article by Elsie King Morland, entitled the "Big Blame".

In January, a member gave a splendid paper on "The Doctor Prescribes Music", telling of the use of music in hospitals, and describing the various kinds of music used and its effects on patients.

In February, Mrs. Gladys Robinson of Welsh, district director of civil defense, and Mr. A. T. Sanders, one of the state directors, spoke to the group on "Civil Defense". Movies were shown.

In March, "Medico-legal Problems" was presented to the auxiliary by their District Attorney.

At the April meeting the Senior and Junior girls from the Jennings and Welsh high schools were honored at a tea. Miss Clare M. H. Wangen, Dean of the College of Nursing at Southwestern Louisiana Institute, spoke on nurse recruitment.

At the May meeting, copies of pictures made by famous artists, depicting scenes from World War II were shown and discussed. These pictures are copies of the "Abbott Collection" which toured the country recently.

RAPIDES PARISH

Mrs. Edwin A. Socola, state president, was key speaker at the Founders Day luncheon of the Woman's Auxiliary to the Rapides Parish Medical Society held recently in Alexandria.

On March 27, the auxiliary entertained at a costume party in observance of "Doctor's Day". The event was held at the Alexandria Golf Country Club, Mrs. Chester Fresh was chairman in charge of arrangements. At the auxiliary's regular March meeting, Mrs. Dewitt Milam of Monroe, was guest speaker and had as her topic "Romance and Research in Medicine". Mrs. Milam is fourth vice-president of the Woman's Auxiliary to the American Medical Society and a past president of the Louisiana State Medical Society Auxiliary.

At the April Meeting of the Rapides Parish Medical Society's Auxiliary Mrs. M. S. Freiman was elected President. Other officers elected were Mrs. J. T. Willis, president-elect; Mrs. P. M. Davis, Jr., recording secretary; Mrs. D. D. Salmon, corresponding secretary; Mrs. D. M. Kingsley, treasurer; Mrs. Clarence Pierson, historian, and Mrs. Milton Hinigman, parliamentarian. The election was held following a report by Mrs. Henry C. Gahagan, chairman of the nominating committee. Installation of officers will take place in May.

OUCHITA PARISH

At a recent meeting of the Woman's Auxiliary to the Ouachita Parish Medical Society, Mrs. Najeeb Klam, presiding, a very interesting talk on "Socialized Medicine — The Picture Today" was given by Mrs. C. U. Johnson. Reports were given by the following committee chairman: Bulletin, Mrs. F. E. Polen; Education and Today's Health, Mrs. D. L. Anderson; Civil Defense and Safety, Mrs. C. B. Flinn; Nurses Scholarship Fund, Mrs. Ralph Talbot, and Community Service, Mrs. Harrell Webster. At the March meeting of the auxiliary, Mrs. Cyril T. Yancey, chairman of the Essay committee, introduced and presented prizes to the three essay contest winners. All three winners are students at Ouachita Parish High School. They are Miss Robbin Wiggins, winner of the first prize, F. H. Reagan, Jr., second prize winner and Miss Elaine Williams, third place winner. Also on the program was Dr. Jack T. Jackson who discussed the Chiropractic Bill which is to be voted on in the next session of the Louisiana legislature. Mr. R. C. White, Jr., talked on the Red Cross and told how it helped him personally while he was a prisoner of war. Following his talk, a very informative and interesting film on the Red Cross and its activities was shown by Mrs. Kenneth L. Weldin of the Red Cross. On March 30, Ouachita Parish Medical Society Auxiliary celebrated "Doctor's Day" in cooperation with other groups throughout the nation. The members en-

tertained the doctors at a semiformal buffet supper at the Country Club. It was brought out that there are 103 doctors in the parish organization. Of these 46 are general practitioners; 12 are surgeons; and 28 are specialists. Also there are 15 resident physicians at Charity hospital and two public health doctors.

ORLEANS PARISH

Woman's Auxiliary to the Orleans Parish Medical Society, in celebration of the 20th observance of Doctor's Day, held open house at eight p.m. at the Orleans Club, March 30. Featured on the program was the presentation of an auxiliary donation by Mrs. Phillips Carter, Doctor's Day chairman, in honor of the members of the Orleans Parish Medical Society. Dr. J. O. Weilbaecher, Jr., received this donation which is given to the American Medical Association Education Foundation Fund, designating the Schools of Medicine of Louisiana State University and Tulane University as recipients. The highlight of the evening was the viewing of a hobby exhibit by members of the Orleans Parish Medical Society. Dr. Branch Aymond was in charge of arrangements.

The winners of the eighth annual National Essay Contest for Junior and Senior High School students were announced at the meeting in April. The contest is sponsored by the Association of American Physicians and Surgeons with the cooperation of county and state medical societies. Mrs. Lloyd J. Kuhn was contest chairman and Mrs. Mannie Mallowitz, co-chairman. The subject for the essay was "Why the Private Practice of Medicine Furnishes this Country with the Finest Medical Care". First place winner in the Senior High School Group was James Wesner of Fortier High. Second place winner was Jules Harris of Holy Cross. Third place went to Mary Arnold of Holy Name of Jesus, and fourth place to Rodney Rue of Fortier High. Miss Loretta Babst was first place winner in the Junior High group. She is a student at Ursuline. Lynne Welsch, McMain High, was second place winner. Third place went to Mary Ann Terranova of Peters High School, and fourth place to Helen Kerr of McMain High.

The students were presented by Mrs. Morgan J. Lyons, Dr. Edwin Zander made the presentation of awards. Mrs. Mannie Mallowitz introduced the contest judges who were Mrs. Alberto Prieto, Miss Henrietta Keitz, Mrs. H. Leon Mintz, Miss Carrie Spurgeon, Mrs. Garland Taylor, Judge Walter Hamlin, Dr. Daniel J. Murphy, and Dr. J. Morgan Lyons. Schools participating and their auxiliary sponsors were as follows: Fortier High, Mrs. Monte Meyer; Warren Easton, Mrs. Frank Oser; Sacred Heart, Mrs. Joseph L. Treadway; Jesuit, Mrs. Phillips Carter; Redemptorist, Mrs. Joseph M. Hountha; McDonogh High, Mrs. John Dunn; Samuel Peters, Mrs. Nicola J. Tessitore; McMain, Mrs. Charles Robinson; St. Joseph Academy, Mrs. Vincent Blandino; Holy Name of Jesus, Mrs. Martin O. Miller; Holy Cross, Mrs. Percy Phillips; Behrman, Mrs. Carroll Gelbke; Ursuline, Mrs. J. Morgan Lyons; and Capdau, Mrs. Spencer B. McNair. First Place winners will compete in the National contest.

Woman's Auxiliary to the Orleans Parish Medical Society celebrated its 25th anniversary at a birthday party held at the Orleans Club, April 14. Mrs. Joseph E. Brierre introduced all the living past presidents. The past-presidents were: Mmes. J. A. Storck, S. M. Blackshear, Isadore Cohn, Francis E. LeJeune, S. Chaille Jamison, Rogers Brewster, W. P. Gardner, Jules Myron Davidson, C. Grenes Cole, Aynaud F. Hebert, James W. Warren, Paul G. LaCroix, C. L. Peacock, John S. Dunn, H. Theodore Simon, Carroll F. Gelbke, Joseph S. D'Antoni, Anees Mogabgab, Edwin S. Socola, Boni deLaureal, John Sanders, Louis Leggie, and Mrs. Edwin R. Guidry, who is now president. A talk on "State Legislation" was given by Mrs. Arthur D. Long, of Baton Rouge.

Presiding at the punch bowls were: Mmes. Spencer McNair, John L. Dileo, William Kohlman, George M. Haik, Vincent Blandino, J. M. Hountha, Monte Meyer, and Harry Morris.

Assisting in the dining room were Mmes: Robert F. Sharp, William B. Clark, Adrian B. Cairns, Philip Boudreaux, Luis R. Oms, Joseph H. Allen, J. W. Warren, Merrill C. Beck, Martin T. Van Studdiford, John B. Gooch, Bruno F. Mancuso, T. A. Soniat, Abe Golden, John H. Dent, R. L. Simmons, Frank G. Nix, William A. Roy, Albert Habeeb, J. D. Russ, Jerome E. Landry, and Daniel C. Riordan.

BOOK REVIEWS

The Calculation of Industrial Disabilities of the Extremities, by Carl O. Rice, Springfield, Illinois, Charles C. Thomas, 1952, Pp. 289, Price $10.50.

This system of evaluating residual disability of the extremities is based entirely on residual joint motion. It has the advantages of simplicity, reliability and a wide scope of applicability. It does have disadvantages in that it does not take into consideration the factors of pain, weakness, and sensory loss and does not take into consideration the type of employment in which the client or patient is involved.

It has been most valuable in the guide or basis for establishing relative disability. It appears that it would be most useful for a physician who makes

only an occasional estimate of disability.

The author has attempted to establish the relative values of the various joints in an extremity in relation to the extremity as a whole and to the body as a whole. Its illustrations are simple, easily understood and anatomically correct. It should prove of value to all who are called upon to perform evaluations of industrial disabilities following injuries.

JACK WICKSTROM, M. D.

Diagnostic and Experimental Methods in Tuberculosis; by Henry Stuart Willis M. A., M. D., F. A. C. P. and Martin Marc Cummings, M. D. F. C. C. P. Springfield, Illinois, Charles C. Thomas, 1952, pp. 394, 43 illustrations, 501 references, $10.00.

This is the second edition of a very useful compilation of methods of value in the diagnosis of tuberculosis. Divided into three sections (Laboratory Diagnosis, Related Clinical and Laboratory Methods, and Some Methods of Value in the Study of Tuberculosis Experimentally Produced), it presents the great majority of those technical procedures which will greatly assist in establishing the tuberculous etiology of a given malady. Throughout the book, the authors endeavor to point out the clinical usefulness of the various procedures and to indicate that one cannot throw the full weight of diagnosis upon the chest roentgenogram.

Doctors Willis and Cummings have done the profession a great service in collating all this material. Because of the diverse appearances of the tubercle bacillus in practically every organ of the body, much of the less commonly employed diagnostic methods are described in specialized tomes devoted to orthopedics, dermatology, neurology, etc. The physician who must frequently diagnose tuberculosis by laboratory methods and the practitioner who only occasionally must resort to this type of study will both find this a very convenient and handy reference volume. The language is simple, the illustrations pertinent and the arrangement logical. *Diagnostic and Experimental Methods* will unquestionably fill a great need in many laboratories.

SYDNEY JACOBS, M. D.

Retinal Circulation in Man and Animals; by I. C. Michaelson, Charles C. Thomas, Springfield, Illinois, 1954 pp. 146, 95 ills., Price $6.75.

A monograph in book form of 146 pages, 95 illustrations and numerous tables. Five phyla of animals are discussed in sole detail regarding embryology, morphology, and physiology of their retinal circulation. Several mammalian animals, especially man, are described. The author freely refers to previous authorities for much information which he himself has not obtained. However, his opinion of anatomical relationships was obtained from flat retinal preparations made by India ink injections. Microscopic sections were also used.

Although much of the material is not original, and with most species, large numbers of animals are not used, the preparations pictured are quite convincing as to the true coursing of the retinal vessels. Photographs and photomicrographs of the actual material used lend authority to the fine detail and careful preparation in this work.

The publishers should be congratulated in lending their support to a work of such basic nature, and issuing it in their usual excellent binding and layout.

J. WILLIAM ROSENTHAL, M. D.

Electrocardiography in Practice; By A. Graybiel, P. D. White, L. Wheeler, C. Williams, W. B. Saunders Co., Philadelphia, 1952.

The authors point out in their preface that this third edition is still primarily an atlas of electrocardiograms for the medical practitioner. Sections are devoted to illustrating the variations in the normal, disorders of rhythm and conduction, changes produced by drugs and chemical agents, and electrocardiographic "patterns" such as specific chamber enlargement, pericarditis, etc. The longest portion in the book is given to records from patients with various etiologic types of heart disease. This is followed by a short collection of "unknowns."

By including the historical summary of the development of physiologic concepts in clinical electrocardiography the atlas has acquired some of the characteristics of a textbook. Minor objections might be raised to the assertion that the Q-T (rather than Q-U) interval is prolonged in hypopotassemia (Figs. 253 and 263) and the use of "myocardial scarring" as an electrocardiographic term (Fig. 271) but the otherwise careful wording of the text emphasizes the individuality of each tracing and the nonspecificity of patterns found under the etiologic types and should thus help avoid the fallacy of matching tracings with the book. The greatest value of the atlas will probably be as a supplement to more complete texts, thus enabling the beginning student of electrocardiography to acquire quickly some of the historical background and to practice reading a variety of well-reproduced tracings in the absence of an extensive practice file.

LEO G. HORAN, M. D.